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THE PRACTICE OF MEDICINE

THE PRACTICE OF MEDICINE

BY

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PREFACE TO THE NINTH EDITION

The work has been very thoroughly revised since the last edition appeared in 1908. Considerable alterations and additions have been made in the subjects of Cholera, Dysentery, Aphasia, Hysteria, Obstruction of the Bronchi, Phthisis, the Physical Examination of the Heart and Vessels, Colitis and Appendicitis, Ankylostoma, Gall-stones, Anæmia, Leuchæmia, Diabetes and Functional Albuminuria; and a short notice of methods of treatment has been included in the Introduction. The subjects new to this edition are Pellagra, Meralgia Paræsthetica, Pneumococcal Meningitis, Myotonia Atrophica, Amyotonia Congenita, Enterospasm, Hirschsprung's Disease, Intermittent Claudication, Congenital Family Cholæmia, Peritoneal Adhesions, Lymphatism, Bacilluria, and Pneumaturia.

The illustrations in the text have been increased to sixty-seven in number; and I have to express my indebtedness and thanks to Dr. Kennedy for the chart of Mediterranean Fever, to Dr. Aldren Turner and Dr. Grainger Stewart for permission to use drawings of nervous diseases, to Dr. Henry Head for the use of seven diagrams of sensory defects, and to Dr. Thomas Lewis for six tracings and electro-cardiograms illustrating the heart's action in disease.

A third edition of the Italian translation by Dr. Luigi Ferrio, of Turin, is in course of publication in two volumes, and the first volume has already appeared.

FREDERICK TAYLOR

August 1911

PREFACE TO FIRST EDITION

I HAVE attempted in this work to offer a short yet complete account of the present state of medical practice, which may be useful both to students and practitioners. I have devoted most attention to the description of Symptoms, to Diagnosis, to Prognosis, and to Treatment, feeling that they are the divisions of the subject which most answer to the idea of practice. Ætiology and Pathology are also of course considered, but the latter could not be so fully dealt with as in works devoted especially to it.

In the arrangement of the diseases, I fear this work may be open to some criticism. Every fresh discovery, every change of opinion as to the pathology of a disease, is likely to call for an alteration in a classification which has essentially a pathological basis. More modern study tends to show that diseases formerly regarded as having a local origin in viscus or joint are of a much more general character. Thus, it is doubtful whether pneumonia, chronic Bright's disease, and gout should not be looked upon as general disorders, rather than as diseases of the lungs, kidneys, and joints respectively. Diabetes mellitus, diabetes insipidus, and hæmoglobinuria, although disorders of the urine, are not due to disease of the kidney; but their true position in classification is still very uncertain, and provisionally they may remain where I have placed them in this book. Similarly, rheumatism and rickets, classified with diseases of bones and joints, are obviously disorders involving a much wider area, but too obscure in their origin to demand a readjustment as yet.

By consulting the most recent works, especially those of Fagge, Strümpell, Payne, Ziegler, Gowers, M. Mackenzie, Douglas Powell, Ralfe, H. Morris, and Crocker, to whom I must express my

indebtedness, I have sought to bring this book fully up to the modern state of knowledge. I have not, however, devoted much space to the discussion of theories, finding that the facts of medicine are amply sufficient to fill, and more than fill, a volume such as this, and being convinced that these facts require to be seized and held fast by the beginners in medicine, not only for the sake of diagnosis and treatment, but also for the right estimation of the various theories which are advanced. With a brief statement, therefore, of such views I have in most cases been content.

FREDERICK TAYLOR

20 Wimpole Street,
Cavendish Square, W.
January 1890

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ERRATUM

Page 847, *for* Methæmoglobinuria and Sulphæmoglobinuria *read* Methæmoglobinæmia and Sulphæmoglobinæmia.

THE PRACTICE OF MEDICINE

INTRODUCTION

A Manual of the Practice of Medicine should deal with Diseases, their Nature, Course, and Treatment.

It has never been very satisfactorily determined what is to be called disease, or what are to be called diseases. To say that disease is any divergence from health, or anything that is the opposite of health, opens up the question, What is health? To this the answer is: A perfect structure of all the organs or tissues, with a perfect performance of all their functions; and, in the broadest sense, any alteration of structure or function may be called disease. But some special cases have to be considered.

A distinction is commonly drawn between injury and disease; but the inflammation which so often results from injury is disease of structure and function, and although the immediate effects of injuries are not generally spoken of as diseases, remote troubles occur which are so classed.

Slight ailments, whether of a structural kind, such as mild catarrhs, or of a functional kind, like headaches, are often spoken of not as diseases, but as disorders, or ailments. There is, however, no essential difference, and no broad line of distinction can be drawn between those which pass off readily, leaving no trace, and those which persist, or recur frequently, or finally shorten life.

There can be no doubt that the structural changes of the various organs and tissues of the body constitute diseases, such as inflammation of the lung or pneumonia, chronic inflammation or cirrhosis of the liver, and cancerous growth of the stomach. Such changes are spoken of as *primary* disease, meaning thereby that each is the first essential lesion in the history of the patient's illness, although each owns some precedent cause, such as contagion by micro-organism in the case of pneumonia, alcoholic indulgence in the case of cirrhosis, and some hitherto unknown causation in the case of cancer of the stomach.

We also cannot deny the name of disease to the disturbances of anatomy that may follow such primary lesions in some cases. Thus, cirrhosis of the liver leads to effusion of liquid into the peritoneal

cavity, abdominal dropsy or ascites; cancer of the stomach, involving the pylorus, causes secondary dilatation of the walls of the stomach; valvular disease of the heart causes congestion of the liver and kidneys, and dropsy of the feet and legs.

Then, again, as a result of either of these groups of anatomical changes, there are certain disturbances of functions, and abnormal subjective sensations, such as pain, vomiting, jaundice, the passage of albumin in the urine, and others. Are they also diseases? There is no difficulty in coming to a conclusion that in these also there is disease.

Lastly, we have to deal with a class of disorders in which no structural change whatever can be found: these are mostly referable to the nervous system; they consist of pain, or spasm of muscle, or loss of sensation, or loss of power, or vascular changes secondary to functional alterations in the vasomotor nerves. This change in every case is for the time being disease, and the different instances are grouped as *functional diseases*.

But the subject may be approached from another point of view. Having decided that the term *disease* has a very comprehensive meaning, we may seek to determine how we shall employ the word *diseases*—that is, how we shall arrange what we know of the possible changes of structure or function into separate groups for practical purposes. These practical purposes, in relation to diseases, are their recognition during life, when the investigation of structure cannot be so complete as after death, and their subsequent treatment. We then perceive that the various changes of structure, which we cannot ourselves see, are accompanied by certain other changes, secondary diseases, or functional disturbances, which are so many indications, or *symptoms*, of the deeper change. Thus, with pleurisy we have pain, cough, and shortness of breath; with cancer of the stomach, pain and vomiting. These indications may be subjective experiences on the part of the patient, like pain, tingling, inability to eat or move; or objective signs to the physician, such as swelling, hardness, elevation of temperature. And among these objective signs must be mentioned especially the indications known as *physical signs*, a term used mostly in reference to the examination of the chest and abdomen with the eye (inspection), the hand (palpation), and the ear (auscultation or percussion). Thus, *symptoms* and *physical signs* are regarded as forming the sum total of all the results of a structural lesion which can be manifest to the physician and guide him in his opinion.

Now, first, the patient has certain experiences of disorder and discomfort, which are to him a very real illness or disease; secondly, the physician, by his special knowledge, can show the existence of other disturbances unperceived by the patient; and, thirdly, there are still facts which he may be unable to observe, and which are only revealed by the pathologist on *post-mortem* examination.

Are we to limit the term diseases to the last or to the second of

this series, or may we allow it to be used also for the headaches, vomiting, dropsies, convulsions, &c., which are the most prominent part of the patient's suffering, but which are to the physician only the symptoms of something deeper, which he is constantly striving to identify? Provisionally, no doubt, we must allow the term a wide application. For, in the first place, there is the whole class of functional disorders, which have, as far as our present knowledge extends, no anatomical basis, and which must, nevertheless, be distinguished from one another and treated; and secondly, there is the fact that lesions of the deeper organs may be very imperfectly revealed even by conditions which cause much suffering to the patient.

But though we may be sometimes compelled to think of dropsy, or headache, or neuralgia as a disease, we must remember that we should never rest content with that position until every effort has been made to find out what organ or structure may be at fault; and we should never permit ourselves to fall into the slovenly habit of ticketing all symptoms with the name of diseases, and treating them without any attempt to remove, or modify, the ultimate cause, when it may be with no great difficulty ascertained. This search after deeper causes, which is incumbent on every physician with regard to the case before him, is being prosecuted with the greatest vigour by the profession in the case of those disorders known as functional diseases; and it is possible that many of them will at no great distance of time be found to be due to structural changes, which at present elude our observation.

A thorough knowledge of every disease requires an acquaintance with several separate branches of study, which are as follows:

Ætiology, the study of its causes in general. *Pathology*, the study of its causes within the body, and the processes resulting therefrom. *Morbid Anatomy*, the alterations in the structures caused by it. *Symptoms*, the indications—subjective on the part of the patient, and objective to the physician—by which we arrive at a knowledge of what is wrong. *Diagnosis*, the method of distingnishing from one another the diseases that may have symptoms more or less nearly alike. *Prognosis*, the art of foretelling the course, duration, and termination of any given case. Finally, *Treatment*, the chief aim of the study of the science and art of medicine.

Ætiology.—The causes of disease are commonly divided into *predisposing* and *exciting*, but no broad line can be drawn between them. A predisposing cause may be in operation for a great length of time without the disease being produced, whereas the exciting cause is usually only of short duration; but conditions which act as predisposing causes at one time may act as exciting causes at another. *Ætiology* strictly covers the whole of the causation of disease, but it is perhaps more often applied to the remoter causes, and to those conditions which are constantly associated with a disease, although we are unable to say how they influence its occurrence. The relations to disease of age, sex, climate, hygienic

surroundings, food, and preceding illnesses are commonly considered under this heading. On the other hand, changes taking place in the body, immediately preceding or causing the disease, are considered rather as pathological processes than ætiological factors.

Pathology is sometimes used to signify the study of diseased structures, but it is better limited to the study of diseased processes—that is, practically, the influence which the causes of disease have upon the function and structures of the body; while **morbid anatomy** or **pathological anatomy** describes in detail the diseased structures themselves.

Symptomatology is the study of the symptoms of any disease; and associated with this and with its morbid anatomy we have to consider what have been called **complications** and **sequelæ.** Complications are certain lesions or symptoms which are the result of the original disease, but only occur from time to time, and are not regarded as a necessary part of the disease; thus, abscesses are a complication of enteric fever; hæmoptysis a common complication of phthisis; parotitis a very rare complication of pneumonia. But the term is applied somewhat arbitrarily; for instance, in enteric fever, rose spots and diarrhœa are not universally present, and yet they are regarded as part of the disease and never as complications. We must regard in a different light the case where one disease occurs at the same time as another, but as far as our own knowledge goes is quite independent of it; the complication may seem to be purely accidental, yet the primary disease may have disposed the individual, in some way not hitherto ascertained, towards the acquirement of the second. Two common cases are (1) the complication of any slight or severe local disorder by an infectious disease, such as scarlet fever, caught by contact; (2) the termination of some chronic nervous disease, like hemiplegia or locomotor ataxy, by pneumonia or bronchitis.

Diagnosis is the study of the recognition of diseases by certain symptoms, physical signs, or facts in the history of the cases which, taken together, indicate that the patient is suffering from a particular ailment.

By *differential diagnosis*—a somewhat tautological term—is meant a knowledge, in any given disease, of all the other diseases which most clearly resemble it, and the points of difference upon which reliance may be placed to distinguish it. But the word *diagnosis* is not always used with the same extent of meaning. Some would limit its use to the cases where, after a careful consideration of the symptoms, signs, and history, aided by their knowledge of the pathological processes to which each organ is liable, they *infer* what they cannot actually see—namely, that the patient has this or that disease. But the term may be extended to the *recognition* of quite obvious lesions, where the amount of inference required is little or none, as, for instance, where one "diagnoses" a black eye, a lacerated wound, a fractured bone with the fragment projecting from the skin, or a pleural effusion after exploring the

chest, and seeing the serum in the syringe. There is, however, no sharp line of distinction between these groups of cases, and they are all of them technically diagnoses. Similarly, it may be said they are all differential diagnoses, since the recognition of a particular disease cannot be considered sure unless the diseases most like it are deliberately, or instinctively, as it were, excluded. Every diagnosis requires obviously a careful consideration of the symptoms, physical signs, and other indications of disease; but, in addition, the former history of the patient and the duration of the symptoms are of the utmost importance; and finally, the physician must have a tolerably complete knowledge of the lesions to which each organ is liable, and of their relative frequency under different conditions. Probability is an important element in diagnosis. In exceptional instances a disease may present a physical sign, symptom, or indication which is not caused by any other known condition. Such a sign or symptom is said to be *pathognomonic* of the disease in question. As a fact, the number of *pathognomonic symptoms* is a very small one.

It will have been gathered from the remarks on diseases that by diagnosis we shall always aim at finding out the *primary* lesion; thus, we must not be content with calling any pain rheumatism or neuralgia, but we must try to find out whether such pain is caused by pressure on a nerve, by inflammation of a nerve, or by degeneration of nerve-tracts. But in many cases the patient suffers from nervous symptoms, pain, cough, sickness, dropsy, albuminuria, and others. As a rule, we should try to see how far all these conditions may be due to one single primary lesion, such as valvular disease of the heart, or granular disease of the kidney; but we must not forgot that frequently two or more independent lesions co-exist, and produce a complex arrangement of symptoms.

Prognosis.—Successful prognosis requires a thorough acquaintance with the natural history of every disease, with the extent to which the disease is influenced by age, sex, and other ætiological factors, and a careful judgment on the variations of the patient from day to day.

The questions that arise in prognosis are such as these: Will the patient recover? Will he recover completely, or be left with any organ damaged? Will he ever have the disease again? If it is a fatal disease, how long will he live?

In the early days of a disease the question of recovery can only be answered by a consideration of the percentage mortality as known by statistics. As the case proceeds, the rapidity or severity of the symptoms, the conditions of the circulation (pulse or heart), the ability to take food, and the integrity of the nervous system, are the points which have most bearing on one's opinion. In practice, prognosis is often of the greatest importance for the physician's credit, and a hasty conclusion, which turns out wrong, is often remembered against him more than any want of success in treatment.

When in this volume it is stated that the prognosis of any disease is favourable, this means, not that it is never fatal, but that *most* cases recover; if any particular symptom or complication renders the prognosis *less* favourable, this means that the percentage mortality of cases with such complication is greater than it was before, or without, the complication.

Treatment.—In this we should aim first at the removal of the cause where this is possible; if not, we may succeed in neutralising its influence. One or other of these methods may suffice to cure all the symptoms and troubles of the patients; but in most cases we are also called upon to deal directly with the symptoms, using remedies that have no influence upon the underlying disease. We must, when doing this, never forget that such symptoms hold a position secondary in importance to that of the disease which causes them. Lastly, we must in all cases counteract the tendency to death, which may, indeed, be the natural course of the disease, or may arise rather as an accident from some infrequent complication. As an example we may take phthisis, which is due primarily to the invasion of the lung by the tubercle-bacillus. The removal of this, when once it has obtained a footing, cannot be directly effected. Its influence can be neutralised by the best hygienic surroundings, by fresh, bracing air, and by special climates which enable the body to resist the action of the bacillus, and possibly by the modification of the patient's tissues by the use of bacterial vaccines. In the meanwhile, there are numerous symptoms—cough, expectoration, pain, sweating, diarrhœa—which will diminish as the condition of the lung improves, and which can be also controlled by suitable medicines In addition, serious complications may arise, especially hæmoptysis, or spitting of blood, by which life is directly threatened, and such a death may be averted by proper therapeutical means. Pleuritic effusion is an instance in which we have cough, dyspnœa, pain, and distress due to the presence of liquid in the pleura; the removal of the liquid either by tapping, or by the use of drugs, is followed by relief of all the symptoms.

It is more than ever necessary to realise that the treatment of an illness does not consist simply in giving internally some form of medicine, but that the processes of the body can be influenced in many other ways. So much is this the case that it seems desirable that a short account should here be given of the therapeutical measures which are available, and which are, of course, described in full detail in works on therapeutics.

Diet.—This has to be considered from the points of view of quantity and of quality. Patients who are confined to their beds as a rule want much less than persons taking active exercise, and in febrile states and many gastric conditions not only is less food desirable, but it must be of the most readily digestible kind. It must also be fluid, since the patient in fever neither has the power to masticate nor the secretion sufficient to insalivate solid food. In

different diseases the relative proportions of the proteid, carbohydrate, and fatty elements of food may have to be altered; thus, proteids are restricted in gouty conditions, and carbohydrates in diabetes. To give rest to the stomach in gastritis, and gastric ulcer or hæmorrhage, feeding by the rectum is often adopted.

Drugs.—The British Pharmacopœia gives the official list of drugs which are required in treatment, but fresh drugs are constantly being introduced, and older drugs not now in the Pharmacopœia may still be of service. Besides the older methods of administration by the mouth and rectum, by friction with ointments, and by medicated baths, drugs are now introduced by hypodermic injection and intramuscular injection; and more recently by *ionisation* or *cataphoresis*. In this method decomposable salts, of which one or both of the component elements are efficient drugs, are applied to the skin and decomposed by a galvanic current, so that the nascent elements or *ions* are driven at once into the tissues, and exert the desired effect.

Organo-therapy.—As a special form of drug, which can be given internally or *per rectum*, or by subcutaneous injection, must be mentioned the extracts of various organs of animals (thyroid, suprarenal gland, testis), which may be made to supply defects in the corresponding organs in the human subject.

Antitoxic and Antibacterial Sera.—These are of great value in some infectious bacterial diseases, especially in diphtheria. They consist of the serum of an animal which has been rendered immune to the particular disease; and the serum contains anti-bodies which will neutralise the disease in the human patient (*see* p. 19). They are standardised by careful experiments upon animals. They are usually injected subcutaneously.

Bacterial Vaccines.—These represent another outcome of the bacteriological study of disease (*see* p. 21). A vaccine is a solution containing several million dead bacteria of the same species as those of the disease requiring treatment, and preferably cultivated from some obtained from the patient to be treated. The injection of these bacteria increases the opsonic power of the patients' blood serum, and thus assists in the ultimate destruction of the bacteria causing the disease.

In the remaining therapeutical methods the forces of nature are applied in various special ways.

Heat.—Besides the local application of warmth for purposes of stimulation or counter-irritation, and of heat for its destructive effects (cautery), *radiant heat*, by means of incandescent electric lights, is employed for the local treatment of rheumatic joints and allied conditions.

Light.—The general advantages of a bright, sunny climate are well known, and even in less clear climates it is of use in some complaints to expose the patient to bright sunlight whenever the opportunity offers. The Finsen light treatment of lupus and rodent ulcers consists in directing an intense light upon the diseased part

for specific periods of time. The light consists of violet and ultra-violet rays, and is produced by an arc-light from which the heat-rays are cut off.

Roentgen rays or X-rays.—The powerful effects of these light-rays are well known, both for good and evil. Constantly playing upon the unprotected skin, as in the case of X-ray operators, they have caused intense and incurable dermatitis. Used with proper precautions for limited periods, they modify the growth of cells in the body, and have been of value in the treatment of rodent ulcer, cancer, enlarged glands, leuchæmic spleen, syringomyelia, ringworm, and other affections. They cause a slow degeneration of the cells, acting more upon the pathological than upon the healthy cells. At a certain point vascular dilatation and extravasation of phagocytes occurs.

Radium.—The rays emanating from this substance have also powerful effects for good and evil. Radium is being used extensively for the treatment of cancer, especially in the deeper passages, where surgery is difficult.

Electricity.—The chief uses of this force have been in the treatment of paralysis and other nervous diseases. Muscles which cannot be stimulated by the will can be made to contract by electric stimulation, so long as their nutrition is normal and they are not the subject of atrophy. This contraction effected at stated intervals maintains the circulation of blood and lymph in the muscles, and facilitates the return to health. Such contractions can be effected by the faradic or by the continuous current. Many painful neuralgic affections are benefited by a continuous current of electricity. Another application of electricity now often used is that of high-frequency currents. These are currents of high potential, perhaps 10,000 volts, alternately positive and negative, and changing their sign about every millionth of a second. They are consequently too rapid to stimulate sensory nerves or motor nerves or muscles, which can only respond to stimuli of about $\frac{1}{1000}$ second duration; nevertheless they have certain effects upon the tissues, which are claimed to be increased cellular activity, changes in the vascular system, and inhibition, *i.e.*, diminished susceptibility of the neuro-muscular system to ordinary stimuli.

Baths and Douches.—By these means heat and cold and mechanical effects may be produced, and so the vasomotor system and the circulation may be affected both locally and generally. Medicated baths, alkaline baths, and sulphur baths are extremely useful in some circumstances, and water giving off carbonic acid in minute bubbles is claimed to have a decided effect even upon the size of the heart's cavities.

Massage.—By this is meant the manipulation of the patient's limbs and body by an operator, in such a way as to assist the circulation of blood and lymph in the part, and to supply the stimulus to metabolism which is furnished in the healthy by active exercise. In the same way stiff joints may be improved, and adhesions

loosened. Locally it is employed after injuries to the limbs, and it is valuable when applied to the body as a whole, in several nervous disorders, such as hysteria and neurasthenia (*see* under Neurasthenia).

Swedish Movements, Nauheim Treatment, Regulated Exercise, Frenkel's Movements.—All these are active movements made by the patient himself under the direction of the physician or operator, with the object of providing a daily amount of exercise either locally or generally, in relation either to the functions of a joint, or the powers of a weak heart, or the cure of obesity, or the working of the digestive functions. Or, as in the case of Frenkel's exercises, the object may be the re-establishment of the function of co-ordination in muscular movements.

Prevention.—The *prevention* or *prophylaxis* of disease will only be occasionally referred to. It is the main object of the study of *Hygiene* or *Public Health*, by which the community endeavours to ward off all external influences adverse to health. But the body itself may be prepared against the operation of the causes of disease, partly by judgment in matters of diet, exercise, clothing, &c., and partly by such special treatment in relation to particular diseases, as is effected by vaccination and antityphoid inoculation.

CLASSIFICATION

Some time ago diseases were divided into *general* and *local*, the latter being those in which particular organs or parts of the body were mainly or alone affected, such as the heart, the lungs, the brain, or the skin; and the former those in which the whole economy was disturbed, so that it could scarcely be said that one organ was more affected than another. The peculiarity of many of these complaints was that they were accompanied by fever, that they were contagious, or transmissible from one person to another, and that one attack protected the individual more or less completely against a subsequent attack.

Improved methods of research with the microscope led to the proof that not only did the *contagium*, or *virus*, or medium of transmission from man to man, consist of visible particles, but that these particles were themselves living organisms, different in different diseases, and capable of cultivation and reproduction both within and without the body. The study of these micro-organisms is the science of *Bacteriology*.

The general diseases were thus seen to be both infectious and specific, and of these typhus, scarlet fever, measles, small-pox, and influenza are examples. But extended researches showed also that many diseases hitherto considered local were also due to specific and infective agents: for instance, pneumonia, tetanus, and diphtheria, as well as suppurative fever; and in their intimate pathology these disorders with their pronounced local manifestations must fall into line with the well-known fevers, such as typhus and small-pox. Classification then is rendered difficult by the fact that if the

organs of the body are taken as a basis, there are *general* diseases which affect the whole body simultaneously and no one organ in particular: but when these are investigated and their bond of union is found to be the infective micro-organisms, then it is seen that a number of diseases affecting mainly one organ or part of the body (hence *local* diseases) must be included with the infectious disorders; and as the tendency of all modern research is to discover micro-organisms as the cause of every inflammatory and degenerative lesion, and even of new growths, the list of infectious diseases is constantly being increased by the addition to its general disorders of diseases hitherto considered local and non-infective.

Within the limits of one physiological system, the same difficulty arises: and the attempt to separate the diseases of the brain from those of the spinal cord, or those of the stomach from those of the intestine, frequently fails, because the parts are simultaneously affected by some common cause. Thus, locomotor ataxy and general paralysis of the insane are both results of syphilis: and several organisms will cause inflammatory lesions at the same time of the brain and spinal cord, of the cerebral and spinal meninges, or of the gastric and intestinal mucous membranes.

There is, however, an advantage in retaining many of the diseases, formerly called local, as long as possible in their old groupings, and it is this, that in their physical signs and symptoms they are necessarily comparable with the other local disorders of the same group, whether the infectious nature of these latter has been shown or not. Consequently the first section on "infectious diseases" will be found to contain those which are obviously general diseases, with some only of those which have pronounced local characteristics. The succeeding sections will deal with the diseases of the various systems, nervous, respiratory, cardiac, alimentary, &c., amongst which it will be admitted that many have an origin in specific infection.

INFECTIOUS DISEASES

NATURE OF INFECTION

By infectious diseases is meant the diseases which depend upon the introduction into the body from without of a *virus* or *contagium;* and this contagion or infective agent, wherever it can be demonstrated, has proved to be a microscopic living being, or *micro-organism,* which can multiply within the body.

The micro-organisms which are related to the infectious fevers require for their detection special methods of staining, as well as very high powers of the microscope. The greater number belong to the class of *Schizomycetæ* or *fission-fungi,* and are called the *lower bacteria.* These are all minute cellular bodies, devoid of a nucleus, and possess the faculty of being stained, when dead and dried, by certain dyes, such as methylene blue, gentian violet, and fuchsin. They occur in various forms, such as minute spherical or ovoid bodies, called *cocci* or *micrococci;* straight rod-like bodies, called *bacilli;* spiral or screw-like bodies, called *spirilla.* Cocci may adhere together in long threads or chains (*streptococcus*), or in plates (*merismopedia*), or in cubical groups (*sarcina*), or in an irregular manner (*staphylococcus*). *Zooglœa* is a term given to masses of cocci or rods united together by a gelatinous intercellular substance. Some bacteria possess *cilia* or *flagella,* by means of which they acquire the power of independent movement. The flagella are few or many, generally longer than the body of the cells and spirally twisted.

These micro organisms are reproduced by *division* and by the *formation of spores* within the bacterial cell (*endogenous*). The first leads to the most rapid multiplication of the organisms, and is spoken of as a *vegetative stage.* The second, or sporulation, takes place under special circumstances in bacilli and some spirilla; growth and multiplication are relatively slow, and the process is regarded as a *resting stage.* Spores are more resistant to destructive agents like heat, drought, and disinfectants than the micro-organism itself.

Another group, the *higher bacteria,* belonging to the class of *Trichomycetæ,* are also sometimes the causes of infectious disease. These are of somewhat greater size, consist of filaments made up of simple cells, and have special organs of reproduction in the cells called *gonidia.* The recognised forms are *beggiatoa, thiothrix, leptothrix, cladothrix,* and *streptothrix.*

A little higher in the scale are *Moulds* or *Hyphomycetæ,* to which the organisms of ringworm and favus belong.

A few infectious diseases are dependent upon organisms which

are admitted to belong to the animal kingdom in the class of *Protozoa*. They are generally less minute, have more variety of structure, and in some cases multiply by a definite sexual process. Examples are the *amœba* of dysentery, the *hæmamœba* of malaria, the *trypanosoma* of sleeping sickness, and probably the organisms of relapsing fever, syphilis, yaws, and kala-azar.

In relation to disease another division has to be made. Some bacteria are proved to be actual causes of disease, and are called *pathogenic bacteria* or *parasites*. They thrive on the living animal and vegetable tissues. Others are not usually the causes of disease; they may be found in association with the parasites; but they flourish in dead and dying animal tissue and in vegetable and inorganic matter; they are called *saprophytes* or *saprophytic bacteria*. However, some saprophytes may become parasites and cause disease; and, conversely, most parasites can thrive on artificial media, and hence behave as saprophytes.

With the first discoveries of pathogenic micro-organisms, it was naturally supposed that the *specific* cause of the disease had been found; but in the sense that each particular organism is peculiar to one disease alone, this has not proved to be true. In some diseases only one pathogenic micro-organism is found, and this may be regarded as *specific, e.g.* in tuberculosis, anthrax, and typhoid fever. In other diseases, such as erysipelas, infective endocarditis, septicæmia and pneumonia, more than one pathogenic organism has been found. The conditions which an organism must fulfil in order to be regarded as pathogenic were first laid down by Koch; increased knowledge required their amplification, and they were thus given by Kanthack. A pathogenic specific germ (*a*) must be a parasite or a facultative parasite; (*b*) it must be found invariably in the tissues of an animal dead from, or affected with, the disease in question; (*c*) it must never under any circumstances occur in other diseases, nor within the normal tissues; (*d*) the organism transmitted from the diseased or dead animal to an affected susceptible animal must reproduce the lesion, and in this second diseased animal the original organism must be found; (*e*) if the organism can be cultivated outside the animal body, then an artificial cultivation inoculated experimentally into a susceptible animal must again produce the disease, and this animal must again contain the organism in its tissues or blood; (*f*) these processes must occur in invariable succession under identical conditions; (*g*) the toxins and poisonous substances obtained from the artificial cultivations must agree chemically and physiologically with those obtained from the diseased animal.

The organisms of a limited number of diseases, including anthrax, diphtheria, and tetanus, fulfil all these conditions; those of glanders, tuberculosis, actinomycosis, gonorrhœa, and malignant œdema fulfil all but the last. Diseases in which there is more than one pathogenic organism have been already mentioned. On the other hand, no specific organisms have been proved hitherto

in rabies, dengue, typhus, mumps, small-pox, chicken-pox, measles and rubella.

Many lesions complicating the above diseases are due to *secondary infection* by the organisms of pus (*streptococcus*, *staphylococcus*, pneumonia (*pneumococcus*), and others.

Action of Contagion in the Recipient.—The virus, or the micro-organism, where such exists, enters the system by the lungs (scarlet fever, typhus, small-pox), the alimentary canal (enteric fever, cholera), the generative mucous membranes (gonorrhœa, syphilis), or by abrasions of the skin (syphilis, hydrophobia). An attack of the corresponding disease does not necessarily follow: for the individual may not be susceptible (*natural* or *acquired immunity*), or the organism may not have the necessary virulence. Streptococci, pneumococci, and diphtheria bacilli have been often found in contact with the tissues of healthy persons.

If, however, the organism is virulent, and the individual susceptible, the entry of the virus is followed by a period of *incubation*, during which no changes are manifest, and which varies generally from two or three to twenty-five days, being generally fairly constant for each particular disease. During the period of incubation the organisms are developing and multiplying, and elaborating the poisonous products to which for the most part the different symptoms and effects of an infectious disease are due. The possible products of bacterial action are many: for instance, gases, fatty acids, bodies of the aromatic series, pigments, ferments, and ptomaines; but the most important of all in reference to disease are the toxalbumins, albumoses, or toxic proteins, which have been found in the fluid in which bacteria have been cultivated, and which have been shown to be the agents to which the symptoms can in most cases be attributed.

It is by the action of the bacteria and their toxins that the greater number of the pathological changes are produced which we know as the basis of disease in the body. These can be only briefly referred to here. They consist of acute local changes both at the seat of inoculation (or entrance of the virus) and elsewhere, whether inflammation, hæmorrhage, œdema or necrosis (vaccinia, syphilis, diphtheria, enteric fever); eruptions on the skin, or *exanthems* (ἐξ out, and ἀνθέω, I blossom), which may be either acute or chronic (scarlatina, measles, syphilis); various chronic local lesions, with cell-growth, such as the so-called infectious granulomata (tubercle, syphilis, actinomycosis); more widely distributed lesions, such as the cloudy swellings of glandular cells, hæmorrhages in various parts of the body; and, lastly, changes in metabolism, which result in malnutrition, cachexia, and often in febrile reaction or *pyrexia*.

The micro-organisms are sometimes confined to the seat of inoculation or invasion, while their poisons or toxins alone are diffused through the system (*toxæmia*); or the micro-organisms multiply in the blood-vessels, and are carried by them to the organs

and tissues (*septicæmia*). In the latter case, they may become impacted in different parts of the circulatory system, and thus form fresh foci of disease. This is seen in the lungs in ordinary *pyæmia*, and in the liver in *portal pyæmia*.

During the progress of the illness, the bacteria or their germs are given off from the patient in various ways, and may thus become a source of infection to other individuals.

The *duration* of a specific disease is often very strictly limited. Thus typhus, relapsing fever, scarlatina, measles, small-pox, and vaccina have all a definite duration, which is rarely more than three weeks, and is adhered to with some constancy. In other acute disorders the duration is longer and more variable, but generally measured by weeks. In others again, as syphilis, leprosy, and tubercle, the infection may be lifelong; but in the first of these there are limitations to the duration of the primary and secondary lesions, which assimilate it closely to the typical specific fevers. How this termination of the infection is brought about—that is, what kills the micro-organisms, or renders their poisons innocuous—has not been clearly shown. The causes which have been suggested, and which are still under discussion, are: the influence of the febrile temperature (*see* Relapsing Fever); the destruction of the bacilli by leucocytes (*phagocytosis*); and the formation in the blood or tissues of substances (*anti-bodies*) prejudicial to the bacilli and their poisons.

Transmission of Infectious Diseases.—This is really a branch of preventive medicine, but a brief notice of it cannot be excluded from a work like this. The infectious diseases having been defined as those in which a virus (or micro-organism) is introduced into the body, it must be here stated that the virus is derived, first, from other human beings ill of the disease, directly or indirectly, as in scarlatina, measles, and many others; or, secondly, from animals, as in rabies, anthrax, foot and mouth disease; or, thirdly, from the soil or other source independent, as far as is known, of the previous participation of other men or animals in the process, as in tetanus. When transmitted from one human being to another, it may be, apart from experimental inoculation, conveyed in solid tissues, in liquid secretions, normal and pathological, in expired air, in clothes, or other articles. In many cases, as in small-pox and diphtheria, the breath appears to be the means by which the poison is conveyed; in others, as in scarlet fever, the skin and secretions from the respiratory mucous membrane; in others, as cholera or enteric fever, the fæcal discharges; in others, like syphilis and glanders, the pus from sores; while in malaria, yellow fever, and sleeping sickness, and perhaps relapsing fever and typhus, the organisms are conveyed and inoculated by mosquitoes or other insects. The exhalations from the breath and skin render the patient *contagious* in the proper sense of the term—that is, that those who are near to the patient for a longer or shorter time run some risk of catching the disease; the fæcal evacuations commonly reproduce the disease by infecting the

water or milk which others drink, or, possibly, the air which others breathe; and lastly, pus containing the virus must come into direct contact either with the mucous membrane or with an abrasion on the surface of the skin.

The period during which a patient suffering from an infectious disease can convey it to others is determined by the duration of the infection in him (*see* p. 14). It begins no doubt with the appearance of the earliest symptoms, that is, at the end of the period of incubation, and in acute diseases is generally limited to three, four, or five weeks. If contagion is conveyed by desquamating skin (scarlet fever), the scabs of pustules (small-pox, varicella), secretions from the throat (diphtheria, scarlet fever), or unhealthy stools (enteric, cholera), the duration will depend on the persistence of these conditions. The practical application of the facts known with regard to these points is illustrated below.

Mixed Infections.—Bacteriological study soon showed that the old doctrine that two infectious disorders could not attack the body at the same time was incorrect, and on the other hand that the occurrence of one infection often rendered the body even more susceptible to a second. Moreover, the virulence of many organisms is an extremely variable quantity, and is dependent in part upon the pre-existing operation of others. Some of the more familiar instances of mixed infections are the co-existence in the same person of scarlatina with diphtheria, of scarlet fever with whooping-cough, of scarlet fever with chicken-pox, of diphtheria with measles, of whooping-cough with broncho-pneumonia, of tubercle with specific pneumonia; but the most important and frequent, perhaps, is the secondary invasion of the body in a great number of infectious diseases by the pus-forming organisms, *staphylococcus pyogenes aureus* and *albus*, and *streptococcus pyogenes*, leading to suppurative lesions, septicæmia, and pyæmia as complications or sequels of the original disease.

Prevention of Infection.—There are three ways by which the transmission of infectious diseases from one person to another, or others, may be prevented. One is by separating the sick from the healthy (*isolation*); another is by destroying the virus in the sick person, or in whatever clothes, books, room, or furniture he may contaminate, or in whatever excreta may pass from him (*disinfection*); the third is by so modifying the condition of the possible recipient that he becomes insusceptible to the influence of the virus, even if brought into contact with it (*production of immunity; immunisation*).

Isolation.—The patient should be placed in a separate room, if possible on a separate floor of the house, which may be screened off by a sheet wetted with solution of carbolic acid (1 in 40). Thorough ventilation must be as far as possible kept up, as the dilution of the poison by a constant influx of fresh air is a most important part of the process. All unnecessary furniture, curtains and carpets, clothes, &c., to which contagion may adhere, should be removed from the room. The attendants should be, as far as

possible, those who are protected by a previous illness; and it should be remembered that their clothes may convey the disease as they pass from the sick room to other parts of the house, unless such overclothing is changed before coming into contact with others. Only such books, papers, or toys should be allowed in the sick room as may be afterwards burned; and food removed from the sick room should not be eaten by other people.

Isolation from susceptible or unprotected persons should be maintained as long as the patient is believed to be infectious. The Medical Officers of Schools Association has adopted the following as the shortest times which should elapse between the appearance of the rash or other commencement and the return of the patient to his home or school: In rubella, ten days; in measles, two weeks; in mumps, three weeks, including one week from the subsidence of all swelling; in diphtheria, four weeks, providing all discharges have ceased and no specific bacilli can be found in the nasal or pharyngeal mucus; in pertussis, five weeks, including two weeks free from spasmodic cough, or whoop; in scarlatina, six weeks, provided desquamation is completed and there is no sore throat, discharge from ear or nose, suppurating gland, or eczematous patch. In small-pox and varicella, all scabs should have fallen off and all sores should be healed.

Disinfection. *Disinfection of the Excreta.*—In enteric fever or cholera the infective agent is contained in the stools. These should be disinfected by thorough mixture with strong carbolic-acid solution (1 in 20), mercury perchloride (1 in 500), or sulphate of iron crystals, and left to stand for two hours before being thrown away. It is better, if practicable, to burn the stools after mixture with sawdust and the addition of turpentine or naphtha; or to destroy them with strong mineral acid, and bury them in the earth. The sputa and urine should be also disinfected in enteric fever.

Disinfection of the Clothing.—Linen may be disinfected by prolonged soaking in solution of carbolic acid before washing. Woollen clothes must be exposed to a dry heat of 180° or 200°, and this is best done in special ovens constructed for the purpose, now in possession of most of the local sanitary authorities.

Disinfection of the Patient.—After the patient has recovered, and before he mixes with his friends, he should have several warm baths and be rubbed with carbolic soap. The special treatment required in scarlet fever, on account of the prolonged desquamation of the epidermis, will be described.

Disinfection of the Room.—After the patient has left the room in which he has been ill, it requires to be thoroughly disinfected before it is occupied by others. This may be done with *formalin* or with *sulphurous acid gas*.

In using formalin, a special apparatus (the Alformant lamp or Lingner's glycoformal apparatus) is required: the room must be securely sealed and exposed to the vapour for at least four hours.

Sulphurous acid gas is obtained by burning sulphur. Two pounds of sulphur should be used for every 1000 cubic feet of space in the room; it is placed in one or more earthenware vessels or pipkins, and each should rest on two or three bricks in a large pan of water. The chinks of the windows should be pasted up with slips of brown paper; the sulphur should be set alight, and the door should be closed and pasted up in the same way as the windows. After twenty-four hours the room may be entered, and the windows thrown wide open. Sulphur has the disadvantage of tarnishing metal work, and injuring pianos, sewing machines, &c., and these should be removed before the fumigation.

After gaseous disinfection the wall paper should be stripped off and burned, the floor and woodwork thoroughly scrubbed with carbolic soap, sanitas, formalin solution (2 per cent), or izal (1 per cent.), and the ceiling whitewashed; or the floor, ceiling, walls, woodwork, and furniture may be thoroughly rubbed with bread, which must be afterwards burnt with all the fragments that drop about.

Notification of Infectious Diseases.—In order to give effect to the above principles, the Notification Act (1889) and the Infectious Diseases (Notification) Extension Act (1899) require that the medical officer of health shall be informed by the medical practitioner in the event of his attending any one of the following diseases: Small-pox, cholera, diphtheria, membranous croup, erysipelas, scarlet fever, typhus, enteric, relapsing, continued, or puerperal fever, or any other made notifiable by the local authority.

Immunity.—Persons who are insusceptible to a particular disease are said to be immune, or to have immunity. Such immunity may be partial or complete, temporary or lifelong, innate or acquired. Of the conditions of *innate immunity* little can be said. Some species of animals are immune towards the diseases from which other species suffer; among different races of men the nearest approach to an innate immunity is that of the negro towards yellow fever. In a given race, however, susceptibility varies very much. It is a fact observed every day, that of a number of persons exposed to the contagion of a particular disease only a certain number will catch the illness; the rest will escape, even though they are not apparently protected by any of the methods to be mentioned below. Moreover, in those who are affected from the same exposure to contagion the disease may present very different degrees of severity. It is not only matter of observation, but has been shown by experiment, that susceptibility to infectious diseases is increased by starvation, fatigue, cold, damp, unsuitable diet, and other conditions unfavourable to the general health; while a more local influence seems to be in operation when pneumonia or bronchitis is succeeded by a tubercular invasion of the lungs. But the working of the law is not always clear; and it is quite certain that the fattest and most healthy-looking children of a family often suffer from, and succumb to, the most violent attacks of scarlatina; while others, apparently

more delicate, may come off with a mild illness. A special susceptibility to acute infectious diseases is noticed in the case of some general disorders, as in diabetes, in women after delivery, and in those who have recently undergone surgical operations; in the last two instances the local wound may be the cause of the increased susceptibility, by providing for the contagion a means of entrance to the body. Another factor in the susceptibility to some diseases is the age of the patient, and this point will be referred to when these diseases are described.

Hereditary disposition may be alluded to here as the converse of innate immunity. Tuberculosis is looked upon as the best example of this occurrence. What is transmitted from parent to child is an undue susceptibility to infection; very rarely, if ever, the actual bacillus. But the possibility of direct infection of the offspring through its surroundings, in cases of apparent heredity, must not be forgotten.

Acquired immunity is that which is imparted in one or more ways to individuals previously susceptible. The most common cause of immunity towards an infectious disease is the fact that the individual has already had the disease. There are relatively few exceptions to the rule that scarlet fever, small-pox, chicken-pox, measles, and other such illnesses do not occur a second time in the same patient. This protection is probably closely related to the conditions which terminate a given infection in those who are suffering; the altered condition of the blood and tissues which destroys the micro-organisms persists afterwards for many years, or a lifetime, and antagonises the influence of any subsequent contagion of the same kind.

In contradistinction to this accidentally-acquired immunity is *artificial immunity*, or the immunity intentionally or purposely acquired by the inoculation of the individual with some substance related to the virus or micro-organism which causes the disease. Artificial immunity may be *active* or *passive*. In *active immunity* the body cells or fluids are themselves stimulated by the inoculation to the production of substances (*antitoxins*) which will neutralise the toxins of the disease anticipated. The substance injected may be the micro-organisms in *living culture*, weakened in virulence or *attenuated*; or it may be the same micro-organisms in their full virulence, but in very small amount; or it may be the dead organisms; or it may be the bacterial products or toxins of the disease without the organisms.

The first of these methods has been employed in the cholera of chickens, in anthrax of sheep, and in the human subject in hydrophobia. The micro-organism or virus may be attenuated by growing in a current of oxygen or of air; by passing through the tissues of an animal; by growing at abnormal temperatures; and by growing in the presence of weak antiseptics. The method employed by Pasteur to prevent hydrophobia is described in the chapter on that disease.

The use of *vaccination* to protect against small-pox (*see* p. 77) may

be regarded as another example, since *vaccinia* (or *cow-pox*) is almost certainly small-pox attenuated by transmission through the cow. Immunity has also been obtained in animals by feeding them with dead cultures of bacteria, or with their toxins.

In *passive immunity* the neutralising, and therefore protecting, substance is supplied from without. A susceptible animal is first rendered immune by repeated injections of the virus of a disease, and the blood-serum of this animal is then injected subcutaneously into the individual it is desired to protect.

Such methods have been used for the prevention of small-pox (*vaccination*), typhoid fever, cholera, plague, and hydrophobia. They have also been employed as cures; that is, the serum of immunised animals has been injected in order to neutralise the toxins of organisms already in the body, and causing symptoms (*serum therapeutics*); as, for instance, in diphtheria, tetanus, pneumonia, and septicæmia.

An important factor in the protection of the individual from bacterial invasion is the process known as *phagocytosis*, or the destruction of the bacteria by the leucocytes and other cells of the body. The chief *phagocytes* are the large uninuclear and polymorphonuclear leucocytes, endothelial cells, and some tissue cells: they are attracted to the bacilli, and this attraction is called *chemiotaxis*.

Polymorphonuclear leucocytes and eosinophiles are classed as *macrophages;* hyaline leucocytes, endothelial cells, and fixed phagocytes are classed as *microphages*. The former are more powerful in dealing with the bacteria of acute disease, the latter with those of chronic infections.

It has been shown that the entrance into the body of toxins, whether contained in the bacteria (*endotoxins*) or produced from bacteria (*exotoxins*), will cause the formation of *antitoxins*. This is only one instance of a large group of similar occurrences, for the injection not only of bacteria and toxins, but of cells, blood-corpuscles, ferments, and other bodies, will cause the formation of *anti bodies*—that is, of substances which act adversely to and destroy the bacteria, cells, and other substances which have been injected.

Another instance is the formation of the bodies known as *agglutinins*. If in certain diseases (enteric fever, Mediterranean fever, dysentery, cholera) the blood-serum of a patient or convalescent is mixed with cultures of the organism of the same disease, in a short time the bacilli are seen under the microscope to lose all active movements, and to become densely aggregated together (*agglutination*, or *clumping*). The same effect may be obvious to the naked eye if the serum and a culture fluid be mixed in a test-tube, when after a time precipitation of the bacilli takes place, leaving the upper part of the fluid clear (*sedimentation*). The results vary with the time employed and with the extent of dilution of the serum. These facts form the basis of the diagnostic method known as *Widal's test* (*see* p. 52).

Agglutinins may also be obtained artificially: for the blood of an animal inoculated with sublethal doses of a given bacillus will acquire *agglutinative* properties towards the bacillus which has been injected (Bordet-Durham reaction).

Precipitins are similar substances, developed in the serum of animals which have been inoculated with bacterial culture-fluids, albumose, milk, &c. The serum containing them precipitates the corresponding culture-fluid, or a solution of the corresponding organic substance which has been used for inoculation.

The actions of *anti-bodies* in regard to bacteria, blood-corpuscles, leucocytes, kidney cells, and other animal cells are known as *bacteriolysis, hæmolysis,* or *cytolysis.* They are for the most part specific, as in the former instance; that is, if certain bacteria are inoculated into an animal the serum subsequently has a destructive effect on the same kind of bacteria only; if rabbit's blood corpuscles are injected into a guinea-pig, the guinea-pig's serum will afterwards dissolve (or lake) rabbit's blood outside the body; if the liver-cells are inoculated the serum will dissolve liver-cells, and so on. These different forms of cytolysis and hæmolysis are dependent not only upon the anti-body produced in the process, but they also require the assistance of another body which exists normally in the serum, and has been called the *complement.* Its presence is shown by the fact that the blood-dissolving power of a hæmolytic guinea-pig's serum may be neutralised by a temperature of 55° to 60° C., which destroys the complement; but it can be restored by the addition of serum from a healthy guinea-pig. The complement is probably a product of the leucocyte, and is identical with the *cytase* of Metchnikoff and the *alexine* of Buchner.

The facts connected with hæmolysis and the action of the complement are utilised in some important diagnostic methods (*see* Syphilis).

Another element in the protective power of the blood is the existence of *opsonins,* bodies which act on the bacteria so that they are more readily digested by the leucocytes (*opsono,* I cater for, or prepare food). The opsonic power of the serum is measured by the number of bacilli which the phagocytic leucocytes can take up (Wright and Douglas).

If a small quantity of blood be mixed with an emulsion of a known bacillus or micro-organism on a slide, placed in an incubator for fifteen to thirty minutes, and a film be prepared from this, fixed and stained, the leucocytes will be found to have included the bacteria, and the average number of these in each leucocyte can be estimated. In different conditions of disease the power of the patient's serum to influence phagocytosis of particular bacilli can be compared with that of the healthy serum, and an estimate of the patient's power of resistance to these bacilli can be formed. For instance, if tubercle bacilli mixed and incubated with washed leucocytes, and the serum of a patient under suspicion, are found to the number of three in each leucocyte, and the same arrangement

with the serum of a healthy person shows an average of four to each leucocyte, the opsonic power of the patient's serum is to that of the normal as 3 : 4 ; or the *opsonic index* is $\frac{3}{4}$, or ·75.

The opsonins combine with the bacteria, and are in turn used up ; but the opsonic power of the serum towards particular bacteria can be increased by the injection of *vaccines* containing dead cultures of such bacteria in measured quantities, the effect being watched by observations upon the leucocytes. The injection of such vaccines guarded by the opsonic index has thus become a definite method of treatment for certain specific infections.

For further information about immunity, and for the theories by which it is attempted to explain it, I must refer the reader to works on Bacteriology.

Classification.—The satisfactory classification of the infectious diseases is impossible as long as the specific organisms of so many are unknown. But even did we know them there are no features which distinguish the diseases caused by cocci from those caused by bacilli ; or those caused by fission-fungi from those caused by protozoa. The attempt to group them according to the organs attacked soon fails. It is true that in some diseases one organ or system is chiefly involved, as the nervous system in tetanus, hydrophobia, and beri-beri ; the bowel in cholera, enteric fever, and dysentery ; and the skin in exanthems : but even in these the frequent "complications" show a specific invasion of other parts, and in many infectious diseases like influenza, pyæmia, and diphtheria, the numerous lesions entirely baffle the attempt at their classification on this basis. The following is a mere summary of facts stated more fully under the separate diseases.

Diseases of more or less certain bacteriology.—Due to Schizomycetes : Micrococci—Septicæmia, pyæmia, erysipelas, cerebro-spinal fever, Mediterranean fever, rheumatic fever, gonorrhœa. Bacilli—Enteric fever, bacterial dysentery, diphtheria, cholera, plague, influenza, tubercle, leprosy, tetanus, glanders, anthrax.

Due to Streptothrix : Actinomycosis.

Due to Protozoa : Amœbic dysentery, malarial fevers, sleeping sickness, kala-azar, relapsing fever, syphilis.

Diseases of uncertain bacteriology.—Typhus, Weil's disease, scarlet fever, measles, rubella, variola, vaccinia, varicella, mumps, whooping-cough, dengue, beri-beri, yellow fever, hydrophobia, foot-and-mouth disease.

PYREXIA

The terms fever and pyrexia are not always used in the same sense, pyrexia being sometimes limited to the mere fact that the body temperature is elevated, while by fever is understood the rise of temperature, together with all the other bodily disturbances which usually accompany it. As is well known, the temperature of the body varies in health between 97·5° and 99° Fahrenheit (36·4° and 37·2° C.). It is usual to speak of 98·4° as being the normal

temperature, and it is very common to find the thermometer give such a record. But there are daily fluctuations of the normal temperature according to which it is lowest between 2 A.M. and 7 A.M., gradually rises from 7 A.M. to 1 or 2 in the afternoon, remains at a maximum from that time to 7 or 8 in the evening, and then falls to its minimum after midnight.

Registration of Temperature.—The temperature of the body is taken, for ordinary clinical purposes, by means of the clinical mercurial thermometer, which registers the temperature after it is removed from the body, by a portion of the mercurial column being prevented returning into the bulb of the instrument. It is usually about four inches long, so that it can be carried in the pocket. The bulb of the instrument may be placed in the axilla, the groin, the mouth, or the rectum.

In the two former situations it is necessary to see that there is complete contact of the skin with the bulb, and it must remain there sufficiently long for the surface of the skin to attain the temperature of the body generally: from one to three or five minutes suffice, according to the sensitiveness of the thermometer. In the mouth the bulb should be placed under the tongue, and the stem must be grasped by the lips. Under certain circumstances the rectum may be employed for taking the temperature; the bulb is introduced for one and a half inches. The result can be depended on; but it is obviously a method that is not always convenient. It may be well to bear in mind another way of ascertaining the body-heat—that is, by passing urine on to the bulb of the thermometer, but its application is rather limited. As a fact, the axilla and the mouth are most frequently employed. Besides the ordinary clinical thermometer, there is in use a thermometer shaped like a very small watch, which is sensitive, but has the disadvantage of not fixing its record; and there are surface thermometers, which are useful in special investigations.

In consequence of daily variations, both in health and disease, it is desirable to record the temperatures at least twice a day: the best times would be 5 or 6 A.M. and 5 or 6 P.M., so that the lowest and highest temperatures should be observed. Social arrangements do not usually allow of this in slight cases of illness, so that 10 A.M. and 6 to 9 P.M. are more often the times selected; but it must be remembered that at 10 A.M. the temperature is already rising, and that after 7 P.M. the maximum is generally passed. In severe illnesses, like typhoid fever, pneumonia, &c., the temperature should be taken at least every four hours, so that the daily variations may be more closely watched; and it should in all cases be recorded on a *chart*, with a dot for each observation and lines drawn from dot to dot. By this means the oscillations of the temperature are graphically represented, its general behaviour is more clearly brought to the mind than by long columns of figures, and a comprehensive grasp is obtained of the course of the fever so far as it can be known from the observations taken. Seeing that the

temperature may not follow a uniform course, but oscillate, even during a four-hour period, it may in exceptional cases be necessary to take it at shorter intervals. When studying the four-hour chart of a long illness, it is often useful to draw up from it a fresh chart, selecting the lowest morning and the highest evening temperatures, and thus showing, in the most prominent way, the extreme oscillation in the body-heat on each day.

Range of Temperature.—In disease the temperature ranges from 93·3° (35·5° C.), or even lower, to 110° or 111° (43·8° C.). Temperatures of 116° and 122° have been recorded, but considerable doubt attaches to their genuineness.

Many terms have been used to denote the different degrees of temperature above or below normal; Wunderlich gives eleven in all, but I think the following are all that are practically wanted:

Collapsé temperatures	92·3° (33·5° C.) or lower, to 96° (35·5° C.).
Subnormal „	96° to 97·5° (36·4° C.).
Normal „	97·5° to 99° (37·2° C.).
Slight or moderate pyrexia	from 99° to 101° (38·3° C.) in the morning, or 102·5° (39° C.) in the evening.
Severe pyrexia	from 101° to 103° (39·4° C.) in the morning, or 105° (40·5° C.) in the evening.
Hyperpyrexia	above 105°.

CONDITIONS ASSOCIATED WITH PYREXIA

Pyrexia, fever, or febrile reaction is accompanied by many other disturbances besides elevation of temperature; indeed, every function of the body is more or less disturbed whenever the temperature is raised for more than a very short time. That this is in part at least a direct result of the high temperature can be shown by experiment; but in nearly all cases of its production by disease it must be recognised that toxins are circulating in the body, and that they are probably the cause of numerous conditions formerly attributed to the high temperature alone.

Skin.—It is hot to the touch, sometimes intensely so; and generally dry, but it may be moist. In some diseases profuse sweats may occur, which sometimes perceptibly, sometimes scarcely at all, reduce the temperature. Such perspirations may cause an eruption of sudamina, or miliaria. The colour of the skin over the body is generally normal, unless there are eruptions, such as miliaria, or the specific rashes of scarlatina, measles, typhus, and others. But the face is often flushed, especially at the commencement of a fever: often the cheeks and lips are flushed, and the face is elsewhere pale; later on, with a failing circulation, the face becomes deeply congested or livid, and the extremities show the same change.

The petechiæ and subcutaneous hæmorrhages which occur in the most malignant forms of infectious disorder (small-pox, typhus, measles, scarlatina) result, no doubt, from the action of virulent toxins upon the capillary walls.

Alimentary System.—The tongue becomes furred; generally, at first, the fur is white, and the tongue is still moist; then the tongue becomes dry, the fur peels from the edges or tip, and shows the bright red tongue beneath. Later on, the tongue becomes very dry, stiff, hard, dirty brown in colour, fissured on the surface, and caked with dried remains of saliva, buccal secretions, food, and sometimes blood mixed with epithelium, fungoid growths and bacteria, which are allowed to accumulate in the passive state of the organs of mastication. In this stage also the gums are covered with a similar collection, which is called *sordes*. Loss of appetite, or anorexia, is one of the first signs of fever; sometimes sickness is present, and in all cases digestion is feeble. The bowels are usually constipated. The spleen is often slightly, in certain diseases very much, enlarged and tender.

Circulation.—The heart's action is quickened, at first excited, then feebler. The pulse ranges from 80 to 120 or more. It is at first full, bounding and firm; it soon becomes softer and dicrotic; and with high fever it is hyperdicrotic (*see* the Pulse). In later stages, as the heart becomes more feeble, it is quick, very small, very compressible, running or flickering. With progressive weakening of the heart's action, the first sound becomes faint, or inaudible, and the impulse may be detected outside the nipple, showing that the heart is becoming dilated.

Respiration.—This is quickened in proportion to the pulse, and the rise of temperature: it may rise to 30 or 40 in the minute. When the illness has lasted some time, the bases of the lungs become congested (hypostatic congestion), and the respiratory movements of the upper part of the chest in front are exaggerated.

Kidneys.—In consequence of the loss of aqueous vapour through the skin and lungs, and of the low arterial pressure, the urine in fever is scanty: and as a direct result of this it is high-coloured and deposits a brick-red sediment of urates on cooling. The urea is in excess, as are the potassium salts, the chlorides are usually diminished, and in some instances the urine is definitely toxic. In severe febrile illnesses there may be a small quantity of albumin.

Nervous System.—Headache is common at the commencement of pyrexia; there is also a heavy feeling, dulness, or disinclination to think or make any mental effort; after a time, the patient not only does not wish to, but cannot exert the intellect; he becomes drowsy, and when he drops off to sleep begins to talk. Later on he is delirious without really sleeping, and the delirium may be muttering, and is occasionally maniacal, the patient getting out of bed, struggling with his nurses or attendants, or jumping out of the window. In the last stages there is profound unconsciousness or coma. In the earlier stage of coma, the patient frequently picks with his fingers at the bedclothes, or catches at imaginary objects in the air in front of him (*floccitatio, carphology*). The disturbance of the muscular system shows itself in general bodily weakness, tremor of tongue or limbs when they are moved, and twitching of the muscles

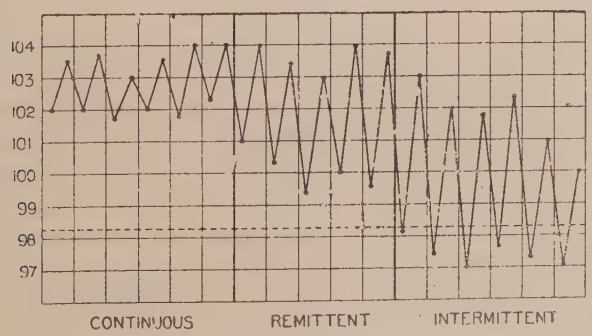
(*subsultus tendinum*), while the relaxation of the sphincter ani allows the uncontrolled passage (incontinence) of fæces, and the diminished visceral sensations lead to retention of urine and dangerous distension of the bladder.

Daily Variations.—The temperature in fever shows daily fluctuations, which are, as a rule, similar to those observed in health, that is to say, the temperature is lower in the morning, and higher in the evening; the lowest point is commonly reached about midnight or 2 A.M., and the highest from 4 to 6 P.M. Occasionally the reverse obtains, the temperature is highest in the morning, lowest in the evening—*typus inversus*. The pulse and respiration rise and fall with the temperature, and the general discomfort of the patient varies in the same manner.

Varieties of Pyrexia.—The pyrexia which accompanies or constitutes an illness may be one of three kinds: it may be *continuous, remittent,* or *intermittent* (Fig. 1). A continuous fever is one in which the temperature is constantly above the normal, and the differences between the morning and evening temperatures never exceed the variation in health—that is, one and a half degrees. A remittent fever is also one in which the temperature is constantly above the normal, but the difference exceeds one and a half degrees. Thus, in the morning it may fall two or three degrees, but never reaches the normal. An intermittent pyrexia is one in which the morning temperature falls to the normal, or below it, while the evening rise is two, three, or more degrees above it. This last form is sometimes called *hectic fever*.

In some cases of illness there may be, at one time, a continuous pyrexia, at another a remittent, and at a third time an intermittent pyrexia, as, for instance, in enteric fever and in phthisis.

FIG. 1



Types of Pyrexia.

Course of Pyrexia.—In many cases of fever a very definite course may be recognised, the beginning and the end being marked by certain characteristic phenomena.

The fever begins with a sensation of cold—it may be a mere

chilly feeling, so that the patient seeks the fire, and he may feel as if cold water were running down his back; or he shudders or shivers, and finally he may have a definite *rigor*. This is an attack of shivering, in which the patient trembles all over, his teeth chatter, he feels intensely cold, his face is pinched, and the nose, ears, and finger-tips are livid. But though the surface is cold, the internal parts are hot, and if the thermometer is used it will be found that the temperature is constantly rising from the first. The rigor may last from a few minutes to half an hour, an hour or more. In young children rigors do not generally occur, but their place is sometimes taken by a convulsion.

The second stage of the fever is the *fastigium*, in which the skin is hot, and the various phenomena already recorded are present.

In some fevers the temperature may rise to its maximum or *acme* just before the onset of the third stage.

The third stage is that of *defervescence* or decline, which occurs either by *crisis* or *lysis*. In *crisis* there is a rapid fall of temperature to the normal within twelve to thirty-six hours; it is sometimes accompanied by profuse sweating, sometimes by diarrhœa (critical sweat or diarrhœa). In *lysis* the temperature falls more slowly, taking three or four days to reach the normal.

For some days after a pyrexia, the temperature may be unusually low (subnormal), *e.g.*, 97° or 96° in the morning, and from this time the period of *convalescence* commences.

Rigor, fastigium, and crisis occur typically in some forms of malaria, and may then all be completed within a period of six to twelve hours.

Death in Fevers.—This results, first, either in a few days, from the virulence of the toxins acting upon the nervous system and involving all the functions of the body, or from their continued action over a longer period; secondly, from hyperpyrexia in a small number of instances only, though it is not uncommon for the temperature to rise very high when the patient is at the point of death from other causes; thirdly, from cardiac failure; fourthly, from pulmonary complications.

Certain anatomical changes are common to nearly all deaths in high fever. The blood is thin, unduly liquid, dark in colour, and rapidly stains the aorta and large vessels. The red corpuscles are diminished, and leucocytosis is common. Small petechiæ or hæmorrhages are found under the serous membranes of the pleura and pericardium. The solid organs—liver, spleen, and kidneys—are large and soft, and the kidneys and liver show, under the microscope, cloudy swelling with some granular change or fatty degeneration in their secreting cells. In hyperpyrexia, the cells of the central nervous system stain diffusely, and Nissl's granules are absent.

The muscles may be soft and friable, or may show the degeneration which was first described by Zenker, and is now regarded as a coagulative necrosis. In this condition the muscular fibres are

converted into a homogeneous, colourless, waxy-looking material, forming cylinders, which break up into fragments, and finally crumble into a granular detritus. This change is most common in the adductors of the thighs, and in the recti abdominis; and it is sometimes accompanied with hæmorrhage, or the broken-down muscle may form a kind of abscess.

The Cause of Pyrexia.—The maintenance of the temperature of the body at its normal standard is dependent upon two factors, *Heat-production* or *Thermogenesis* and *Heat-loss* or *Thermolysis*. If the temperature of the body is above the normal standard, heat-production must be in excess: if below, the difference must be in favour of heat-loss. Heat-production results from chemical changes, of which it is said that 80 per cent. take place in the muscles, independent of their contraction, and controlled by nerves arising from centres in or near the corpus striatum. Heat-loss occurs from the skin to the extent of 77 per cent., by the lungs to the extent of 20 per cent., and the remainder by the urine and fæces. The nerve apparatus concerned is chiefly the vaso-motor system, by which the circulation through the skin is affected. If the skin becomes hot the vessels dilate, the skin is congested, it becomes a better conductor, more blood passes through it, and so heat escapes by radiation to the surrounding air. With high fever also, respiration is quickened, and the loss of heat from the pulmonary surface is proportionately increased. The centre controlling the operation of heat-loss is situate in the medulla oblongata.

Obviously, the temperature will rise if the heat-production increases with normal heat-loss, or if the heat-loss is less with normal heat-production. Pyrexia was attributed by Traube to the latter alone; by others it has been accounted for by the former alone.

It is believed that a regulating function also exists, which maintains the balance between production and loss. It is called *Thermotaxis*, and probably has its seat in the cortex of the brain (Hale White, Ott). In different degrees of fever, according to MacAlister, all three functions may be disturbed. Heat-regulation, the highest function, is disturbed first, producing slight irregular pyrexia; in a higher degree of pyrexia, heat-production and heat-regulation are both upset, the former being increased; and in hyperpyrexia, heat-loss, the lowest function, is also involved. Burdon-Sanderson regarded variations of temperature as depending especially upon heat-loss and upon its regulation, while heat-production is, according to him, always liable to be in excess of actual requirements, so that disturbances of regulation nearly always result in pyrexia and not in collapse.

The next point is to determine how these functions are disturbed —that is, what are the remoter causes of pyrexia? Some of these causes are well known. The groups of febrile disorders usually recognised are (1) infectious diseases, or *specific fevers;* (2) fevers

occasioned by local inflammation, formerly called *symptomatic fevers*; and (3) fevers which arise from local disease, or are associated with even functional failure, of the nervous system: the last have been spoken of as *neurotic fevers*.

To take the third group first: it has been shown by experiment and by cases of disease that lesions involving the supposed heat-regulating centre (cortex cerebri), or the heat-producing centre (nucleus caudatus), or the fibres proceeding thence to the muscles, may be followed by pyrexia of a marked kind. It is seen, for instance, occasionally in cases of cerebral hæmorrhage, cerebral tumour, and meningitis. This is less often a continuous pyrexia than an occasional rise of temperature. The high temperatures caused by peripheral irritation, such as the passage of gall-stones, may be attributed to stimulation through afferent nerves of the same centres, and reflex action there operating.

There are other febrile disturbances for which no local cause can be found, nor any association with the groups which follow. These are commonly regarded as functional, hysterical, or *neurotic*, and are attributed to the failure of the thermotaxic mechanism. Besides being very variable in duration, they are often unaccompanied by the disturbances characteristic of fever, such as anorexia, furred tongue, and loss of flesh.

The *specific fevers* are, as already stated, dependent upon the entrance into the body of bacteria, or micro-organisms, which multiply in the blood or tissues: and the pyrexia must be regarded as due to the operation of the bacterial *toxins* upon the heat-centres or the heat-apparatus. Some other poisons, vegetable and animal, also determine changes in the temperature of the body.

The pyrexia associated with local inflammation (*symptomatic fever*) is probably sometimes due to peripheral irritation, but in later stages—for instance, when suppuration has occurred—it is no doubt due to the absorption of the toxins of the micro-organisms (streptococci, staphylococci) concerned.

Subnormal Temperatures.—The subject of abnormally low temperatures cannot properly be separated from the consideration of pyrexia, and the following list of the causes of subnormal temperatures, given by Janssen, may be found useful: (1) Direct withdrawal of heat from the body, as in cases of exposure of unconscious or drunken persons, in very cold atmosphere, or of immersion in very cold water. (2) Loss of great quantities of fluid from the body, as in severe diarrhœa, cholera, enteritis, or profuse hæmorrhage. (3) Conditions of cachexia and inanition, such as cancer of the various parts of the alimentary canal, severe diabetes, pernicious anæmia, convalescence from febrile affections, and many chronic mental diseases. (4) Grave circulatory disturbances, as cardiac failure and stenosis of the respiratory passages. (5) Various diseases of the central nervous system, tubercular meningitis, the onset of cerebral hæmorrhage and embolism, some cases of cerebral tumour, and general paralysis of the insane. (6) Irritation of

sensory nerves, as in intestinal strangulation, renal and hepatic colic, perforations of the intestine, and surgical operations. (7) Extensive skin affections, such as universal eczema, and large burns. (8) After fevers, when the temperature may long remain subnormal, or in the course of pyæmia. (9) Poisoning by phosphorus, atropine, morphine, carbolic acid, and alcohol; uræmia and diabetic coma.

General Treatment of Diseases attended with Pyrexia.—The treatment of each particular case will depend more or less upon the cause; but the general principles of treatment are as follows: The patient should be at rest in bed, in a well-ventilated apartment; and he should be watched day and night, preferably by trained nurses, and should be kept scrupulously clean. He should also be kept cool, the amount of clothes being lessened if the fever is very high. A distinct lowering of temperature may be sometimes effected in this way, a point to be remembered all the more as the tendency of the patient's friends is to heap clothes upon him to prevent his "catching cold." The extremities, however, must be carefully watched, and specially covered or warmed if necessary. The diet should be light, digestible, and nutritious. In the majority of cases milk is the most suitable, and an adult patient may take from three to four pints in the twenty-four hours; beef-tea, mutton-broth, chicken- or veal-broth, may generally be substituted to the extent of half or one pint in twenty-four hours, and arrowroot, jelly, cornflour blanc-mange, or custard may sometimes be added. In cases where milk disagrees, or is felt to load the stomach, or is rejected, it may be mixed with half its bulk, or an equal quantity, of barley-water or soda-water; or it may be peptonised or pre-digested by warming for a little time with liquor pancreaticus. These foods should be given in small quantities at short intervals; for instance, five ounces of milk every two hours night and day in severe cases. The thirst may be met by soda-water, barley-water, lemonade, toast and water, or cold weak tea; and there is generally no objection to the patient drinking frequently.

In some cases of pyrexia the temperature may be directly dealt with by methods known as *antipyretic*. It must be distinctly understood that such treatment will not lessen the duration of the illness; that in many illnesses the temperature will of itself fall in a few hours, that is, in the early morning; and that there is very rarely (except in rheumatic hyperpyrexia) any danger that it will rise to a height which can be directly fatal. But efficient antipyretic treatment increases the comfort of the patient during the time that each successive dose or application is in operation, and possibly, in some diseases, diminishes the risk of damage to the viscera; and although such a treatment to a certain extent interferes with the natural course of the temperature, it need not materially falsify our estimate of the progress of the illness.

Antipyretic methods may be divided into three groups:

Milder Refrigerants.—These are the ordinary saline remedies

—citrate of potassium, acetate of ammonium, dilute acids, which were formerly given in every fever, but have very little influence.

Stronger Antipyretic Drugs.—If one of these is given in a single dose to a patient with a temperature of 103° or 104°, the temperature falls within two or three hours to normal or even lower, but it rises again in six or seven hours to a height not much different, if at all, from what it would have reached had no antipyretic been given. The following are the drugs which have been most often employed, and their doses: Quinine sulphate, 20 to 30 grains; salicin, 30 grains; salicylic acid, 20 grains; antipyrin (phenazone), 15 grains; antifebrin (acetanilide), 2 to 5 grains; phenacetin, 5 to 10 grains. The last three are the most certain in their antipyretic action, but care must be taken not to exceed the maximum doses here stated, as these drugs are apt, especially antifebrin and phenacetin, to cause cyanosis and collapse when taken in too large quantities. Antipyrin occasionally produces an erythematous or urticarial rash, which is of little importance. Antipyrin has been shown to act by increasing the discharge of heat from the surface of the body, doubtless by an influence upon the nervous centres presiding over the inhibitory or dilating vascular nerves. These drugs are very little employed at the present time for their purely antipyretic action.

External Application of Cold.—This may be done in several ways: The cold bath; the wet pack; sponging; ice applications; Leiter's coils. Though more troublesome than the administration of drugs, its use can be better controlled, and there is less risk of harm to the patient.

Cold Bath.—This has been largely used in the treatment of enteric fever. The temperature is taken every three hours, and whenever it is found at any of these periodical observations to be 102° F. or higher, the patient is placed in a bath of a temperature of 70° F., in which he remains for ten or fifteen minutes, according to its effect upon him. He is then removed, lightly dried, and replaced in bed. The temperature will then be generally found to have fallen to 99°, 98°, or even lower. The system is open to modifications; the observations may be made less frequently, the bath may be only used when the body-heat is 102·5°, or 103°, or 103·5°, and the temperature of the bath may be as low as 60° or as high as 80° or 90°. Sometimes the patient is put in the bath at a temperature of 90°, and ice is then introduced to bring down the heat to 75° or 70°. It is obvious that the greater the number of baths, and the lower their temperature, the greater will be their effect upon the mean body-heat. I have generally made observations every three hours and used a bath of 80°, when the temperature was found at 103°. Continuous immersion has been also successfully employed.

Wet Pack.—A sheet is wrung out of ice-cold water and wrapped round the patient for ten or fifteen minutes, the application being made under the same conditions of bodily temperature as are directed for the bath.

Sponging.—The body is uncovered and sponged over with cold or ice-cold water for from seven to ten or fifteen minutes. This method is not generally so effective as the two former; the temperature commonly falls from one and a half to two degrees.

Ice-bags.—These may be placed on the chest or abdomen for varying periods, or hung within a cradle placed over the patient.

Leiter's Coils.—These are closely-wound coils of metal tubing, through which a continuous stream of cold water is passed. They may be applied to the head, chest, or abdomen.

Stimulants.—In all severe febrile illnesses the necessity for temporary stimulation may arise; this may be supplied by sal volatile, carbonate of ammonium, or small doses of ether; but more certainly by *brandy* or other distilled spirit. Signs of nerve-prostration or cardiac failure are the indications for the use of stimulants—quick, feeble pulse, inaudible first sound of the heart, dry, tremulous tongue, or delirium. A pulse under 100 rarely requires stimulants. If the heart becomes irregular or shows evidence of dilatation, digitalis and ammonia or strychnine should also be given. The quantity of brandy may be from two to six or eight ounces in the twenty-four hours; but the larger quantities must not be continued for many days, and, especially in prolonged illnesses like enteric fever, one should carefully watch the effects of this drug, since an excessive amount will keep up a quick pulse, and a drowsy, muttering delirium, deceptively like the very condition for which it was originally given.

Strychnine is also a valuable drug under similar conditions: two or three minims of the pharmacopœial solution may be injected subcutaneously once or twice daily.

TYPHUS FEVER

(*Spotted Fever, Putrid Fever, Jail Fever*)

Typhus fever is an acute specific contagious disease, lasting two or three weeks, and producing a typical eruption on the skin.

Ætiology.—Typhus occurs, for the most part, in epidemics, which may last for some months, and then gradually subside. These epidemics commonly break out in large towns, attacking especially the poorer quarters, where overcrowding, filth, and insufficient food appear to act as predisposing causes of its spread, rendering those subject to such conditions extremely susceptible to the influence of the poison, as well as especially fit to further its development. It has also, at different times, raged in prisons and in armies in the field. It is conveyed by clothes and other similar articles, but is not carried by water, milk, or food.

It attacks persons of all ages, and males and females equally. Those who have already gone through an attack are, with rare exceptions, protected from another. It is not much affected by season or weather, except in so far as they may determine over-

crowding; but it is confined to temperate and cold climates. It is at present comparatively rare in England.

Nicolle and Conseil, of the Institut Pasteur in Tunis, have shown that the infection is conveyed by body-lice. They successfully inoculated monkeys with blood from typhus patients, and then by means of lice feeding upon the first monkeys, produced the disease in others. Observations of a recent epidemic in Tunis were in support of the above view.

Symptoms and Course.—The period of *incubation* is variable; in a few instances it has been two days or less; but in more cases it is twelve, thirteen, or fourteen days. The disease begins, like many other fevers, with headache, loss of appetite, and a general feeling of illness, with perhaps some chilliness or actual rigor. In severe cases there are sharp rigors, with nausea, or sickness. During the next two or three days, while yet there is nothing distinctive of the disease, all the symptoms of severe fever are rapidly developed. The temperature rises to 103°, 104°, or 105°, the pulse and breathing are proportionately quickened; there are furred tongue, continued headache, flushed face, and suffused eyes; pains in the back and limbs, anorexia, scanty high-coloured urine, and constipation. By the third or fourth day the patient is generally so ill as to be obliged to take to his bed. On the fourth or fifth day, sometimes as early as the third, sometimes on the sixth or even the seventh, appears the characteristic eruption, or *mulberry* rash, of typhus. It comes out on the abdomen and chest and on the backs of the hands and wrists, and in the course of two or three days covers the trunk, and perhaps also the arms and legs. The face and neck are mostly free. It consists of two portions: one, a dusky red mottling, fading on pressure, not giving rise to any elevation of the surface, and often described as "subcuticular"; the other, a rash, consisting of numerous paler or darker pink or red prominences or papules scattered irregularly over the surface, at intervals of one-third to half an inch from each other. These at first fade on pressure, but after a day or two they become more dusky, and later some of them become petechial from extravasation of blood, which persists under the pressure of the finger. The rash gradually fades during the second week, by the end of which it is generally gone. In rare cases the mulberry rash is preceded by a diffused red rash, or *roseola*, not unlike the eruption of scarlatina; but this disappears entirely before the mulberry rash comes out.

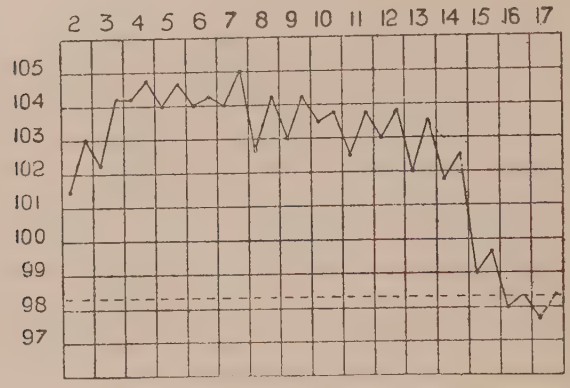
By the time the eruption is developed—that is, at the end of the first week—the fever has made progress. The patient lies on his back in bed, with a dull, heavy, stupid look, the face flushed, the conjunctivæ injected, and the pupils contracted. The temperature, pulse, and respiration are, of course, above the normal; the tongue continues furred. He may still complain of headache, but is mostly apathetic and listless, and only towards night has a little rambling delirium. In the second week the symptoms are aggravated. The headache, indeed, is no more complained of,

but the delirium becomes constant day and night; and, though generally low, and muttering, and accompanied with floccitatio, it is sometimes noisy and raving, so that the patient may become violent, may start from his bed, and even jump out of the window. But more often his muscular weakness is extreme; he sinks down in the bed, and is unable to raise or turn himself; the limbs are tremulous, and subsultus tendinum is observed. Later on, the patient becomes completely comatose, the fæces are passed unconsciously, and the bladder becomes distended from retention of the urine. The pulse is rapid, reaching 110, 120, or more, and with the progress of the illness it becomes feebler and smaller. It is dicrotic, and, in severe cases, may be irregular and intermittent. The sounds of the heart are faint, and in the worst cases the first sound is almost inaudible and the pulse imperceptible. The breathing is rapid, reaching 30 or 40 per minute, and there may be some bronchitic rhonchi over the chest. In the later stages there is generally evidence of congestion of the bases of the lungs —viz., dulness for three or four fingers' breadths from the base of the chest, deficient breath-sounds, and râles over the same area, while the breath sounds under the clavicles are supplementary. The tongue, which is furred at the commencement of the illness, soon becomes dry, brown, and cracked; and the tongue, teeth, and lips become covered with a brown or black crust of sordes. The bowels are often constipated, but there may be diarrhœa; and in either case the motions are generally dark. The urine continues dark and scanty, the chlorides are reduced to a very small amount, and, in a certain proportion of cases, a trace of albumin appears, especially towards the end of the illness. An examination of the blood shows that the red corpuscles are somewhat diminished in number; there is a very slight leucocytosis, with a relative increase of the uninuclears and a diminution of the polymorphonuclears (*see* Diseases of the Blood).

The temperature generally rises on the first day to a considerable height, 103° or 104°, and, continuing elevated, reaches a maximum of 104·5°, 105°, or even 106°, about the end of the first week. It then remains for some days about the same level, with perhaps slight morning remissions. It is commonly a little lower in the second week, towards the end of which it shows more decided morning remissions, and finally, in cases that recover, occurs the rapid fall which constitutes the *crisis* of the disease. This happens in the large majority of cases about the end of the second week, or from the thirteenth to the sixteenth day (Fig. 2). The temperature then falls four, five or six degrees in the course of twenty-four to thirty-six hours, from 103°, it may be, or higher, to 99°, 98°, or lower still. At the same time, the pulse and respiration become slower, and the crisis is not uncommonly accompanied by other indications of change in the condition of the patient—viz., profuse sweating or an attack of diarrhœa. From this time the recovery of the patient is rapid; he soon

regains consciousness, the tongue cleans, the dusky hue of the face subsides, and thirst is replaced by appetite. The temperature commonly remains normal during convalescence, but the pulse is often rapid, 90 to 100, for some days after the crisis. Though at first excessively weak, and continuing to emaciate even after the

FIG. 2



Temperature in a Case of Typhus Fever.

crisis for a day or two, the patient quickly gains strength and flesh, and may be, in the course of a month or six weeks, better and stouter than he was before the fever.

But the termination is often unfavourable; in fatal cases, death takes place commonly towards the end of the second week from cardiac failure, or from congestion of the lungs, or pneumonia, or from increasing coma. It is occasionally preceded by a rapid rise of temperature.

Morbid Anatomy.—The *post-mortem* appearances are scarcely distinctive, but are such as are characteristic of severe fever. The *rigor mortis* is imperfect, decomposition sets in early, and there is much *post-mortem* discoloration; the blood is more than usually liquid, coagulating rapidly but imperfectly. The voluntary muscles are soft and friable, and may undergo Zenker's degeneration (*see* p. 26); the muscular tissue of the heart is also soft, and affected with fatty or granular degeneration. The bases of the lungs are in a state of hypostatic congestion; they are dark red or purple, congested, airless, and friable, yielding blood and serum on section, and without the granular surface of pneumonic hepatisation. This last condition is, however, also present in some cases. The spleen and liver are soft and somewhat enlarged, and the kidneys are often soft and large, though sometimes quite normal. The enlargement of the liver and kidneys is associated with cloudy swelling and parenchymatous degeneration. No definite micro-organism has yet been demonstrated as the cause of typhus fever.

Among the **Complications and Sequelæ** are pneumonia, which often arises during the second week, and persists into the period of convalescence, delaying recovery, and sometimes going on to gangrene; bed-sores; gangrene of the fingers, toes, nose, or pudenda, probably from embolism; suppuration of the joints; inflammation and suppuration of the parotid, submaxillary and sublingual glands; and erysipelas of the face. Thrombosis of the femoral vein, with resulting œdema of the leg, may occur, but is less frequent than in enteric fever. Meningitis has been found in a few instances *post mortem*, and uræmic convulsions sometimes occur, in association with renal disease and albuminuria, which are either of old date or have been set up by the typhus fever itself. Mania sometimes occurs during convalescence.

Diagnosis.—At the outset of the disease it may be impossible to distinguish it from other febrile illnesses, unless it is known that the patient has been exposed to contagion. When the eruption appears it may be mistaken for *measles*, but the rash of measles generally comes out first on the face, near the scalp, the spots are brighter red, more raised, more irregular in shape, and perhaps arranged in crescentic forms, and their appearance is preceded by catarrh. The eruption should be sufficient to distinguish it from *acute meningitis* and from *acute pneumonia*, with which typhus may be mistaken, on account of pronounced cerebral symptoms in some cases, or of respiratory distress and lividity in others. Pneumonia, however, should be recognised by its physical signs, but may, of course, be secondary to typhus. In its early stages, typhus may be mistaken for *enteric fever* (or *typhoid*). The papules of typhus may present resemblances to those of typhoid, but their early appearance, and their occurrence on the fore-arms and wrists, should prevent errors; as well as the general mottling, if pronounced, and the petechiæ in later stages. Other points of difference are the sudden onset of illness in typhus; the absence of diarrhœa in typhus as contrasted with the loose, ochrey, offensive stools which characterise enteric fever; and the early stupor and delirium of typhus. Typhus also spreads rapidly, so that many members of a family may be affected at the same time. Exceptionally, a case of *malignant endocarditis*, with petechial eruption, may closely resemble typhus. If the Widal test be employed (*see* pp. 19, 52) the serum will fail to agglutinate typhoid bacilli, or paratyphoid bacilli, or the micrococci of Malta fever.

Prognosis.—The mortality of typhus is stated to be about 10 per cent., but it varies much with age and other conditions. In children under ten years of age it is about 5 per cent., and in people over sixty years of age it is as much as 66 per cent.; so that age is a very important point for consideration in the prognosis. The chances of recovery are lessened by previous intemperance and by deficiency of bodily vigour from any cause, whether it be overwork, starvation, or overcrowding; even the attempt to keep about during the first days of the attack renders

the case less promising. The symptoms of the illness itself, which suggest an unfavourable prognosis, are abundant rash, very high fever, very rapid pulse, early development of cerebral symptoms, great weakness of the circulation, severe pulmonary complications, and convulsions.

Treatment.—It must be at once understood that there is no known method of cutting short an attack of typhus fever, and that the object of treatment is to maintain the strength of the patient, so as to bring him safely through his illness. To this end he must be dealt with on the principles laid down under the head of Pyrexia (*see* p. 29). Most cases in children, and mild cases in adults, may do without stimulants; but cases presenting the features which suggest an unfavourable prognosis mostly require them at some time or other. Thus, in patients over middle age, in those who have led intemperate lives, and in those suffering from much prostration, they will be wanted early; and they must be at once prescribed if there is much feebleness of the heart and pulse, cyanosis, serious pulmonary complication, much low delirium, muscular tremor, or sleeplessness. Maniacal delirium, however, seems to be aggravated by their use. From four to six ounces of brandy may be given in twenty-four hours, in divided doses; but very severe cases may require as much as ten or twelve ounces. The bladder should be constantly watched; its over-distension will probably result in the frequent passage of small quantities of urine; and in this case the catheter should be used at once. If there is much headache, sleeplessness, or delirium, cold may be applied to the head by means of an ice-bag or Leiter's coils; or opium may be given as tincture or the liquor opii sedativus; or morphia may be injected subcutaneously; thus, a grain of opium or a quarter of a grain of morphia may be given at night in the first week. Chloral in a dose of ten or fifteen grains is more suitable when the delirium is maniacal, and it may be combined with the same quantity of potassium bromide. For sleeplessness, paraldehyde (℥ss to ℥j in water), or sulphonal (twenty grains) may also be used. In the later stages of the disease, sedatives are less desirable, and are contra-indicated by coma, severe lung complications, and suppression of urine. For lung complications, carbonate of ammonium is the best remedy; and turpentine is also of value. When the temperature is very high, sponging the body with tepid water will often give temporary relief to the sensations of discomfort which the patient suffers; but the systematic employment of cold baths to reduce the temperature, such as have been used in enteric fever, has not been found to give very satisfactory results. On the third day of convalescence, if the tongue be clean, solid food may be given; and the stimulants required in the height of the fever should be gradually diminished.

Prevention.—If the disease is habitually conveyed by lice, the destruction of these insects, and the fumigation or destruction of the clothes and bedding which harbour them, should be the

means of prevention. Conseil recommends the fumes of sulphur, as superior to formalin, in destroying both the insect and the virus.

RELAPSING FEVER

(*Famine Fever, Febris recurrens*)

Relapsing fever is a specific contagious disease, generally occurring in epidemics, not distinguished by any rash, but consisting of a short fever which terminates suddenly in six or seven days, and is followed by a relapse of the same nature after an interval of about a week.

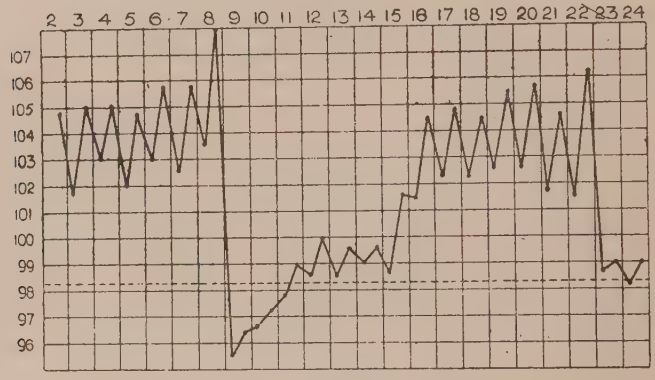
The micro-organism of relapsing fever is a spiral body which was first discovered in the blood by Obermeier in 1873, and was called *spirochæta Obermeieri;* but it has been shown by Schaudinn that it is really a phase of a trypanosome, possessing a large nucleus and a micro-nucleus, and altering its shape, so as to become an oval or pear-shaped body, with a large and small nucleus. As usually seen it consists of an exceedingly fine thread, from 16μ to 40μ in length by 1μ in width; it is spirally coiled, and in constant movement of a rotatory or lashing character. Spirilla may adhere to one another so as to form masses, or they adhere, either singly or many, in a tufted manner to the blood-corpuscles. Their quantity in the blood varies with the different stages of the illness. They are always present during the paroxysms, first appearing, according to some, about the second day; according to others, in the stage of incubation for forty-eight hours before the onset of fever. They frequently increase in numbers as the fever progresses, but about the time of the highest temperature, just preceding the crisis, begin to diminish; by the termination of the crisis they have completely disappeared from the blood, and they remain absent until near the advent of the relapse. Both men and monkeys have been infected by inoculation with the blood of patients suffering from the disease. In monkeys affected with this disease, Metchnikoff discovered that when the spirilla disappear from the blood, they can be found in the spleen, for the most part included within, and being destroyed by, the polymorphonuclear leucocytes (*phagocytosis*). But some of the cells are undergoing degeneration, and it is suggested that spirilla thus escaping may again multiply and cause the relapse. The blood also often contains large protoplasmic masses, and free granules.

Ætiology.—Relapsing fever attacks patients of all ages, and of both sexes, though male patients have been more numerous, in the proportion of three to two. The occurrence of epidemics is very little affected by season or weather, but has an obvious connection with overcrowding, destitution, and starvation; and to the last fact it owed a former name, "Famine Fever." Thus hawkers, beggars, and tramps are frequent among those affected by this disease; but of people in a better position, those only are affected

who are brought into direct contact with the disease in hospital or elsewhere, such as doctors, nurses, and clergymen. Thus the disease is distinctly contagious, even amongst healthy people, but the conditions of destitution and starvation are powerful predisposing causes. The spirillum is conveyed from patient to patient by the bite of the bed-bug, or of ticks in some countries.

Symptoms and Course.—The period of *incubation* varies from

FIG. 3



Temperature in Relapsing Fever.

one to sixteen days, but in a large proportion of the cases examined for this purpose, it was less than nine days. The fever begins suddenly with a chill or rigor, soon followed by frontal headache, and pains in the back and limbs. After a short time the chill gives place to a feeling of heat, the skin becomes dry and burning, and the headache and pains in the limbs are aggravated. On the first day the temperature may be 102·5° or 103°, or higher. The patient soon takes to his bed, and suffers severe thirst, with anorexia, and perhaps nausea and vomiting, the face is flushed, and the tongue is covered with a thick white fur. The temperature continues high, reaching 104°, 105°, or 106° at night, though often falling a degree in the morning.

There may be with this an occasional rigor, and not infrequently free sweating. The pulse is rapid, 110 to 120, and the respirations are quickened to 30 or more. In some cases there is jaundice, which may be very pronounced: in such cases the urine is stained with bile, but the fæces are normal in colour. There is frequently tenderness over the hepatic and splenic regions, and both liver and spleen are enlarged, the latter more often and more decidedly than the former. Occasionally there is well-marked herpes of the lips.

The urine is very variable, its quantity being affected by food, vomiting, or sweating. In the height of the fever the chlorides are diminished, and there is occasionally a small quantity of albumin.

Epistaxis sometimes occurs. And though there is no typical rash, in rare cases an eruption has been observed, either of pink maculæ or of petechiæ.

The condition remains much the same for about a week; the patient, as a rule, gets but little sleep, suffers severely from the muscular and arthritic pains, and is generally quite clear mentally, till towards the end, when he becomes delirious. All the febrile conditions then become greatly aggravated, the pulse quickens to 130, the respirations to 40, the temperature rises in a few hours to 106°, 107°, or even 108°, the face is flushed, the tongue dry, or brown, and the delirium is increased; and then suddenly the crisis takes place. Perspiration breaks out, rapidly becomes profuse, and the temperature, pulse, and respiration quickly fall. In a few hours the temperature is 98°, 97°, or 96° (Fig. 3), the pulse 70 or less, the skin is moist, the tongue clean, and the patient free from delirium; and except that he feels weak, may express himself as being comparatively well. However, where the fall of temperature is considerable—and it may be 10° or 12° in twelve hours—and the sweating is profuse, there may be considerable collapse for some hours, especially in elderly patients. The crisis is sometimes marked by diarrhœa or by epistaxis. Recovery from this point is very rapid; the temperature, which has become subnormal, regains the normal level; the patient is soon ravenously hungry, and recovers strength so quickly as to be up in three or four days. He appears, indeed, completely convalescent, when suddenly, about the fourteenth day, or a week from the termination of his first attack, he is seized again with chills and pyrexia, and the whole series of phenomena is repeated. He has the same high temperature, the pains in the head, back, and limbs, and the sleeplessness; and again, after a few days, occurs a second crisis, with the profuse sweating and the sudden cessation of fever. In some cases a second relapse occurs, and in some even a third, fourth, or fifth. But these last form a very small proportion of the cases. On the other hand, some patients escape without any relapse at all. Most commonly the relapse is of somewhat shorter duration than the first attack, lasting on an average four to five days, but it may be only two or three days. It is often milder than the first attack, but it may be more severe; and, indeed, a certain proportion of deaths takes place during the relapse. Sometimes it is indicated only by slight rise of pulse and temperature, and general malaise. Convalescence from relapsing fever is often very slow. The disease is much less fatal than either typhus or enteric fever, the mortality, according to Murchison, being 4 per cent. Death occurs most commonly at the height of the first attack, or immediately after the crisis, from exhaustion and collapse; and this is especially the case with old people. But in some epidemics suppression of urine and convulsions have preceded death; and a fatal result may follow such complications or sequelæ as pneumonia, dysentery, or parotitis.

Complications and Sequelæ.—An important complication is *pneumonia*, which has been frequent in some epidemics, and may be the cause of death; it may be associated with pleurisy, and rarely gangrene of the lung has resulted. The spleen may attain a great size, and it has been known to rupture, with a fatal result. Diarrhœa and dysentery sometimes occur in a severe form. *Jaundice* occurs in probably less than 20 per cent. of the cases, and may appear in the first attack alone, in the relapse alone, or in both paroxysms. Many of the cases in which it occurs are severe, or even fatal, but others are quite mild. In the severer forms it may be accompanied by epigastric and hypochondriac pain, vomiting of blood, albuminuria, hæmorrhages, delirium, coma, and subsultus.

Erysipelas and œdema of the legs occasionally occur, and sometimes the parotid or submaxillary gland inflames or suppurates. *Ophthalmia* occurred in some epidemics, commencing in the deeper structures of the eye, so that blindness was observed before the external signs of inflammation. Pregnant women almost invariably abort, and in such cases hæmorrhage from the uterus may become a danger.

Morbid Anatomy.—With the exception of the condition of the blood, already described, there is no specific or constant lesion. The enlarged spleen, which is especially likely to be found when death takes place during a paroxysm, sometimes presents infarctions, and, more rarely, small abscesses. The liver also is usually found enlarged, firm, and loaded with blood; but the jaundice is not always explained by any alteration of its structure, or by obvious obstruction of its duct. The kidneys may be congested. Other pathological conditions have been alluded to in the description of the complications.

Diagnosis.—In the course of an epidemic, the sudden severe onset, absence of eruption, severe pains in the limbs, and jaundice, when present, are distinctive; but in isolated cases the disease may be confounded with typhus, enteric fever, small-pox, rheumatism, or pneumonia, and in tropical countries with remittent malarial or with yellow fever. Typhus and small-pox are soon excluded by the absence of eruption, but in other cases diagnosis may be difficult until the occurrence of the typical crisis. Even then a latent pneumonia may be thought to explain the case, but the occurrence of a relapse a week later will make it clear. There is often a general resemblance to acute rheumatism in the flushed face, white furred tongue, sweating, and severe pains; but the existence of pain in the muscles and the absence of swelling in the joints should prevent a mistake. In any case, during the febrile period short of the fastigium, the diagnosis may be at once established by an examination of a blood film with a high power, when the spirochæta will be seen.

Treatment.—Recently it has been stated that relapsing fever, in common with all other diseases due to spirilla, is at once arrested

by the new remedy for syphilis proposed by Ehrlich, and known as "606" or Ehrlich-Hata: this is dioxy-diamido-arseno-benzol, of which the sodium salt is injected into muscle or vein in a dose of ·2 to ·3 gramme.

Apart from this, the disease must be treated like other fevers, in the way indicated under typhus. The recumbent position, a well-ventilated room, and fluid food are essential. Sponging with tepid water, or packing in wet sheets, will give temporary relief when the fever is very high, and headache may be treated with cold applications. On the other hand, if there is much tenderness of the liver or spleen, fomentations give relief. During the severe perspiration of the crisis, the patient must be kept as far as possible dry; and the tendency to collapse must be met by additional bed-clothing, hot bottles, and diffusible stimulants. Cardiac failure must be met by the administration of digitalis, or strychnia. The pains in the limbs may be so severe as to require the use of opium or of morphia injected subcutaneously. In the latter part of the interval the patient may take solid food, and tonics (iron, quinine, nux vomica, or mineral acids) may be usefully given.

ENTERIC FEVER

(*Typhoid Fever*)

Enteric fever is a specific disease, infectious chiefly through the excretions. It has a febrile period of about three weeks' duration, and occasionally one or more relapses of the same length; its typical features are an eruption of pink spots, and a variable amount of diarrhœa with characteristic motions. The distinctive pathological lesion is inflammation and ulceration of Peyer's patches in the small intestine.

The specific micro-organism of enteric fever is a bacillus discovered by Eberth. It is 2–3μ in length, with rounded ends, and provided with from eight to twelve fine flagella of about twice its length. It bears a close resemblance to the bacillus coli communis, but can be distinguished from it by bacteriological tests. Eberth's bacillus has been found during life in the stools, in the blood, in the urine, in the sputum; and in the pus of abscesses resulting from periostitis and other similar lesions months and even years after the attack. After death it has been found in Peyer's patches, in the mesenteric glands, spleen (abundantly), liver, gall-bladder, kidneys, meninges, bone-marrow, and, rarely, in the lungs and testicles.

Ætiology.—Enteric fever shows little preference for either sex; but age has a marked influence, and the disease is much more frequent amongst young people. The quinquennial period which presents the highest percentage of cases (viz., 27 per cent.) is that between fifteen and twenty years; nearly 50 per cent. of the cases occur between fifteen and twenty-five, and more than 84 per cent.

between five years and thirty (Corfield). The disease does, nevertheless, occur (1 or 2 per cent.) in people over sixty-five years of age. It is more prevalent in the latter part of the year—that is, in the four months August to November, inclusive—and cases are more numerous during hot and dry weather than under the opposite condition. It is not affected by overcrowding, poverty, and destitution, nor by the occupation of the patient, in the same way as typhus and relapsing fever; and it is very doubtful if anything is given off from the body or the breath of the patient to the surrounding air which can convey the disease to another individual. As a rule, doctors, nurses, and students in hospitals do not take enteric fever directly from the patients. The agent of transmission is, in the vast majority of cases, the fæces; and in those not very common instances in which nurses have contracted the disease from their patients, it was probably by direct contact with linen or bed-clothes soiled with the fæcal discharges. But bacilli are found in the urine in some cases, and are constantly present in the pus from bone lesions (*e.g.*, periostitis) which follow typhoid fever; both these secretions may therefore be the means of transmitting the disease. And the presence of bacilli in the sputum, and in the rose spots on the skin, has also a possible bearing on the spread of the disease.

In the last few years it has been shown that those who have had typhoid fever may convey infection to others many years after they have themselves recovered. The history is generally that isolated, otherwise unexplained, cases have occurred among those associated with such a person. If the individual upon whom the repeated cases have thus thrown suspicion is examined, the bacillus of typhoid is found either in the urine, or less frequently in the fæces; or the blood gives the Widal reaction. Such persons are called *typhoid-carriers:* and they may unconsciously infect water, milk, or things they handle. It is believed that the gall-bladder is the seat of retention and multiplication of the bacilli in these cases.

A frequent cause of the spread of typhoid fever in a town or a country district is the contamination of its water-supply by the stools of a single case. There is nearly always sufficient opportunity for such an occurrence in the imperfect means usually employed for the disposal of sewage. In the country, wells used for drinking water may be poisoned in consequence of the soil being saturated with sewage which has leaked from a neighbouring privy or imperfectly constructed cesspool. In one case a well was contaminated by the slops from a laundress's house leaking into it; enteric fever broke out in the houses supplied by the well shortly after the laundress had received some linen soiled by the discharges from a patient with this disease. Where the drinking water is conveyed by pipes, the disease may find an entrance if the pipes by any chance are defective, and if they lie in a porous soil sufficiently close to any collection of sewage, imperfectly confined, which has received any

enteric stools; and a whole reservoir may be infected in the same way. Drinking water is, however, not the only source of danger. Epidemics of enteric fever have been traced to the milk-supply, the probability being that the milk itself has been first infected by being stored in vessels washed with water exposed to contamination by typhoid sewage. Typhoid has also been traced to ice-creams sold in the street; and to oysters, cockles, mussels, and clams, supplied from breeding-areas exposed to sewage contamination, and eaten uncooked. Watercress or celery may be an agent in a similar way.

Imperfect sewage arrangements will lead to contamination not only of the water we drink, but also of the air we breathe. But it is now generally believed that enteric fever is rarely, if ever, conveyed directly by the air, and that the danger of sewage emanations, in regard to this disease, lies only in the probability that the individual who constantly breathes sewer gas will have the bodily resistance so lowered that he will readily develop enteric fever, if acquiring the bacillus in other ways. In the Transvaal war it appears likely that fæcal infection was assisted by sand-storms and by flies (Tooth).

Symptoms and Course.—The period of *incubation* of enteric fever is variable; but many cases in which it has been ascertained have shown it to be about a fortnight, or between ten and fifteen days. In exceptional cases it may be as short as five or as long as twenty-two days. The beginning of the disease is often very little marked. The patient feels ill, depressed, unfit for work; he has headache, pains in the limbs and back, loss of appetite, and perhaps nausea. These may come upon him so that he scarcely knows when they began; but he can frequently fix a day on which he says he first fell ill. Often the headache is severe, and forms the most prominent complaint. There may be diarrhœa in the first few days; sometimes on the first feeling of illness a purgative is taken, and the bowels continue loose. The patient may go about, struggling to do his work, for five or six days, but generally towards the end of the week he is obliged to give up and take to his bed. The temperature has been stated, in the first four or five days of enteric fever, to rise two degrees each evening, and to fall one degree each morning, so that at the end of that period it will have reached 103° or 104°. So many cases escape accurate observation in the early days that it is not always easy to confirm this, but it is certain that in some cases the thermometer may rise on the first evening of illness to 103° or higher. The high level of 103° to 104° once reached, the temperature commonly remains at nearly the same level till the tenth to the fourteenth day—oscillating, however, between morning temperatures of 102° to 103° and evening temperatures of 103° to 104·5°. The pulse is quick, full, soft, and markedly dicrotic. Though in some cases very rapid, it is generally, in relation to the temperature, much slower than in typhus and many other febrile

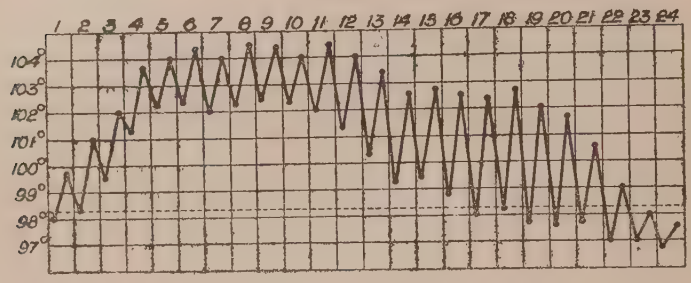
conditions: it may never exceed 100, and a pulse of 80 may co-exist with a temperature of 102° or 103°. The respirations are increased in frequency, and there is very frequently slight bronchitis, indicated by sibilant rhonchi, and accompanied, it may be, by mucous expectoration. About the seventh to the tenth day, the patient commonly begins to present the characteristic appearance of enteric fever. He is dull, listless, apathetic, but not so dull and stupid as in typhus; the eyes brighter, the pupils often dilated; the face pale, with flushed cheeks and dark lips; the tongue dry, with a band of dry white fur on each side, the sides, tip, and middle clean and red. Pain in the head may still continue a prominent symptom, and lead him even to cry out. Occasionally profuse perspiration occurs, or bleeding from the nose. At the end of the first week, or later—that is, from the sixth to the twelfth day—appears the characteristic *rose rash* of enteric fever; it consists of rose-pink spots, circular, slightly raised above the surface, flat, convex, but not pointed, so that they are often described as *lenticular*, from two to four millimetres in diameter, disappearing under firm pressure with the finger, and never petechial like the typhus rash. They are seen first on the abdomen and front of the chest, and may be confined to these parts; but also occur on the sides, back, and the upper arms and thighs. In number they vary from half a dozen to twenty or thirty, but they may be much more numerous, and in a certain number of cases (10–20 per cent.) are entirely absent. Each spot has a limited duration, gradually fading in three or four days; but spots continue to come out day after day until the end of the third week, or in some cases even later. They are not visible after death. In the second week also the *intestinal symptoms* become prominent. The abdomen is generally full, even distended, and resonant on percussion; and there may be both tenderness and pain, but the former is more common than the latter. Pressure in the right iliac fossa, over the seat of the cæcum and lower end of the ileum, often elicits a little pain, and sometimes a sensation to the hand of *gurgling* or of dull crackling, like what is felt when air escapes in the subcutaneous tissue after injuries to the chest (*subcutaneous emphysema*). The examination for this sign must be made with great care, lest injury be done to the diseased bowel. *Diarrhœa* is a characteristic symptom of enteric fever, but it is very variable in duration and in severity. Often there is a sharp attack of diarrhœa in the first week, and after this the bowels are confined; sometimes (up to 40 or 50 per cent. of the cases in some epidemics) there is *constipation* throughout. In other cases diarrhœa is constant, and the motions number three, four, or five or more daily. The stools, moreover, are distinctive in being liquid, of the colour of yellow ochre, and of a peculiar offensive odour. They commonly contain particles of undigested food, intestinal epithelium, bile-pigment, micrococci and bacilli, crystals of ammonio-magnesian phosphate, and after a time shreds of sloughs from the diseased Peyer's patches. They

are alkaline and ammoniacal. The intestinal lesions further show themselves occasionally by the occurrence of *hæmorrhage*. This often happens in the stage of separation of the sloughs or of ulceration, and large quantities of bright red blood are discharged from the bowel, so as to cause severe collapse, with pallor and depression of temperature; but the bleeding may be quite slight, and this more often in earlier stages of the illness. The *spleen* is generally enlarged; this may be manifest only from the results of percussion; but in most cases the organ can be felt, on deep inspiration, one or two inches below the costal margin. The *urine* is scanty, dark, and of high specific gravity; the urea and uric acid are increased, but the sodium chloride is much diminished. Late in the illness albumin is found in a small proportion of cases. But for the headache and some giddiness the cerebral functions may be very little disturbed in mild cases; the headache rarely lasts beyond the tenth day, and there may be then only a little drowsiness or tendency to wander at night. A temporary deafness is not uncommonly noticed. Such mild cases reach their acme at the end of the second week—the tenth to the fourteenth day. The temperature then takes a characteristic course; hitherto standing always at a high level, it now falls every morning quickly lower and lower, while the evening temperatures, though also falling, come down much less rapidly. Thus the morning temperature in four or five days reaches 99° or 98°, while the evening temperature stands at 102° or 101°. This is Liebermeister's *remittent* stage. From this point to the end of the illness the fever has for three or four days an *intermittent* character; it is about normal in the morning, but rises to 101° or more in the evening. Then rather suddenly the evening fever ceases, the temperature remains normal or subnormal; and convalescence has commenced (Fig. 4). During this falling temperature, spots may continue to come out, the spleen is still perceptible, and there may be a little diarrhœa; but the mental condition of the patient generally improves, and he often acquires an appetite some days before the fever has entirely left him.

On the other hand, the graver cases are mostly accompanied by a marked increase in the intensity of the nervous symptoms, to which the symptoms of cardiac failure, or severe abdominal troubles, may be added; more or less continuous delirium may supervene, with drowsiness, or even coma, extreme muscular prostration, subsultus tendinum, and plucking at the bed-clothes. The face becomes dusky, the tongue dry, sordes collect on the lips and teeth, the pulse is rapid, soft and dicrotic, the heart sounds are feeble, and the bases of the lungs are congested, as shown by râles and a very feeble respiratory murmur. The urine may be retained, or both fæces and urine are passed unconsciously. The condition resembles that described under typhus fever: the patient is indeed in a truly *typhoid* state. The delirium is less often violent than in typhus, but occasionally patients get out of bed, or refuse food.

Cardiac failure shows itself by the feebleness of the heart's beat, the indistinctness of the sounds, the small, rapid pulse, and by venous congestion of the face and extremities, and of the bases of the lungs. Sometimes there is evidence of cardiac dilatation in displacement of the impulse outwards, and the pulse may be

FIG. 4



Temperature in Enteric Fever.

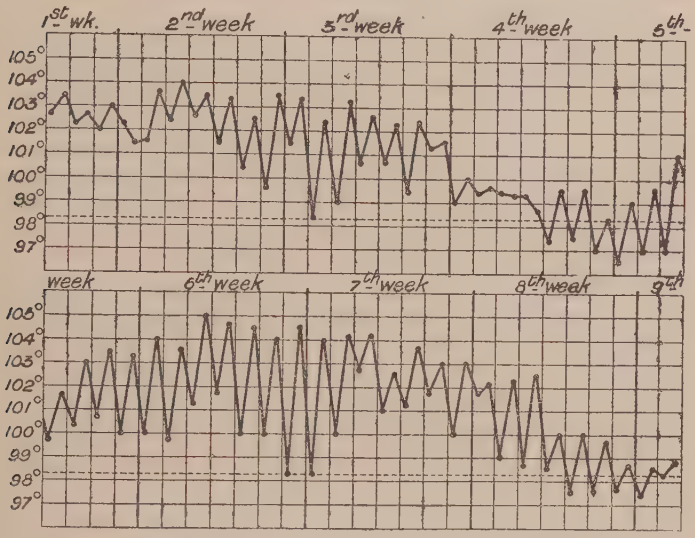
irregular or intermittent. With the increase of the nervous symptoms, the abdominal troubles are often prominent, the diarrhœa becomes profuse, and the abdomen is much distended, tense and tender; in this stage the ulcerated bowel may give way, and peritonitis may result from the escape of fæcal matter into the cavity of the abdomen. In a few cases severe bronchitis is the main feature of the disease; the face is livid, râles and rhonchi are heard over the whole chest, and breathing is seriously obstructed. Under these various circumstances death may occur almost at any time after the tenth or twelfth day; but recovery occurs after lengthened periods of coma and other severe symptoms, the temperature slowly returning to the normal, and convalescence being very protracted.

Relapses.—A true relapse of enteric fever occurs in a certain proportion of cases, which have been found by different observers to be from 3 to 10 or 11 per cent. It consists of a repetition of all the phenomena of the illness, ulceration of Peyer's patches, fever, diarrhœa, and rose spots; and it occurs after an interval which may be as long as eleven days from the termination of the original fever, but is often much less. Sometimes, indeed, there is no interval of actual apyrexia, and the relapse seems to be continuous with the primary fever. Its duration is often quite as long as that of the first attack (Fig. 5), and, as a rule, it is somewhat milder. Death may, indeed, take place in the relapse, but this is more often from complications, such as perforation of the intestine and peritonitis, or from hæmorrhage, than from the severity of the pyrexia alone. Occasionally a second relapse occurs after another interval of apyrexia; and even third and fourth relapses have been observed, though very rarely.

Morbid Anatomy.—The essential lesions of enteric fever occur

in the *Peyer's patches* and *solitary follicles* of the small intestine. These become infiltrated with lymph-corpuscles, and a Peyer's patch so affected swells, and projects one or two lines upon the inner surface of the intestine; it is gray, fawn-coloured or pink, but the surrounding mucous membrane may have its natural colour. The

Fig. 5



Temperature in a Case of Enteric Fever with Relapse.

lymph-corpuscles at first multiply in the follicles, but subsequently infiltrate the mucous membrane above and the deeper structures below. As the patches become larger, they acquire a creamy-white colour, and about the tenth day or a little later they begin to ulcerate, or slough, presenting at first a superficial abrasion at one point of the surface, which becomes deeper and deeper until a great part of the gland is removed; or a whole patch may slough at once. When the slough is still adherent, it often has a yellow colour from absorption of bile. By these processes the muscular coat or the peritoneal covering may be exposed in the floor of the ulcer, and finally, the peritoneum may slough, ulcerate, or tear, so that the contents of the bowel escape into the peritoneal cavity, and set up intense peritonitis. The state of ulceration generally occupies part of the third week, and towards the end of that time, in favourable cases, the process of healing by cicatrisation begins. Ulceration does not necessarily occur. In mild cases the inflammatory swelling subsides without further destructive change. The number of Peyer's patches affected is very variable, and though the cases with severe diarrhœa generally have extensive inflammation of the bowel, there is no necessary correspondence between the extent of ulceration

and the severity of the other symptoms. The patches near the ileo-cæcal valve are those attacked, and the process spreads upwards. The change in the solitary follicles of the lower end of the ileum is of the same kind, and in some cases the lymphoid follicles of the large intestine (mostly the cæcum) are also enlarged and ulcerated. Coincidently with these lymphatic structures of the intestines, the *mesenteric glands* are inflamed; they are enlarged, fleshy, pink, red, or purplish, and their histological changes resemble those of the Peyer's patches. Sometimes they break down so as to contain one or more small collections of fluid resembling pus, which in rare cases burst into the peritoneum; but they may become cheesy or even calcareous. Quite exceptionally fatal cases have occurred in which no intestinal lesion could be found. The *spleen* is commonly enlarged, dark in colour, and, in later stages of the disease, softened. The *liver* is often hyperæmic, and softer than natural; the *kidneys* are congested, and in both these organs the gland cells are granular. The *heart* is often soft and flabby, its muscular fibres being in a state of fatty and granular degeneration. Zenker's degeneration (*see* p. 26) occurs in the voluntary muscles, and was indeed first described in connection with enteric fever. The *lungs* are either œdematous, or congested at the bases; or, in occasional cases, there is actual pneumonia.

Complications.—The complications are numerous and varied, but a large proportion of patients escape them: as might be expected, the most important are those connected with the intestinal lesions.* *Hæmorrhage* (10·21) has been already mentioned (p. 45). *Peritonitis* is a frequent cause of death. It arises most commonly from perforation (3·2) of the floor of one of the ulcerated Peyer's patches, through which the contents of the bowel are extravasated into the peritoneal cavity; but it occasionally happens from extension of inflammation through the peritoneal coat, without any perforation being discovered; and it has also been caused in rare cases by the softening of inflamed mesenteric glands and infarctions in the spleen, and by rupture of the gall-bladder. Perforation of the bowel takes place in more than 30 per cent. of fatal cases of enteric fever. In more than two-thirds of the cases it occurs in the second, third, or fourth week, but it is rare before the ninth day. Its onset is often accompanied by acute pain, collapse, and perhaps vomiting, or rigors; the abdomen is tender, sometimes flat and rigid, at others distended, but in both cases moving scarcely at all on respiration; the pulse is small and rapid, and the temperature sometimes falls. But its advent may only be marked by collapse and increased distension; and in very severe cases, with much distension of the bowel, as well as coma and delirium, there may be no certain signs to indicate peritonitis, so that perforation and peritonitis are occasionally found *post mortem* when not sus-

* The figures in brackets represent the percentage occurrence of each complication among 901 cases treated in the years 1908 and 1909, in the Metropolitan Asylums Board's Hospitals.

pected during life. So long as the ulcer remains unhealed there is a possibility of a rupture taking place; and that such a rupture may be induced by any disturbance of the bowel, as by vomiting, defæcation, the exertion of sitting up, or the administration internally of indigestible food or of aperients; and thus, even cases which are running a mild course may be fatal from this cause.

A slight amount of *bronchitis* is frequent in enteric fever, but occasionally it is so severe as to constitute a very serious complication. The face may be quite livid, and a more or less venous tinge may be given to the whole surface; the chest is filled with râles and rhonchi, and there is expectoration of mucus or muco-pus. *Ulceration of the larynx* occurs sometimes in severe cases. The ulcer is situate commonly over the arytenoid cartilage, and this may be even exposed and in a state of necrosis. Sometimes an abscess forms around the cartilage in consequence of *perichondritis*. As results of these laryngeal complications there may be hoarseness or complete aphonia; subcutaneous emphysema, from air being forced during expiratory efforts from the larynx into the connective tissues; and cicatricial stricture of the glottis in cases that recover. A temporary aphonia may occur without any evidence of ulceration. *Pneumonia* (2·0), sometimes being gangrenous, *broncho-pneumonia* (·5), and *pleurisy* (·88), both serous and purulent, occasionally occur; and much more rarely *pneumo-thorax*. *Jaundice* is of rather rare occurrence: it is not always readily explained; but may be related to the now well-established affinity of the bacillus for the gall-bladder. The stools are not necessarily deprived of bile-pigment; and recovery may take place without any further indications. In some cases local diseases independent of enteric fever cause the jaundice. *Cholecystitis*, or inflammation of the gall-bladder, is a result of direct infection of that organ. *Acute nephritis* (1·55), sometimes with abundant albuminuria or hæmaturia, may occur. In about one-fourth of the cases of enteric fever the bacilli are found in the urine, especially in the third week; sometimes they are so abundant as to cause a visible deposit (*bacilluria*), and sometimes their presence is accompanied by *cystitis*. They may persist there for years, as has been stated in connection with typhoid-carriers (*see* p. 42). *Otitis* (4·5) and *otorrhœa* may occur during or after the fever, and may lead to deafness, or to the more serious conditions of *septicæmia* and *meningitis*. Nevertheless, meningitis is quite rare as a complication of enteric fever, and the cerebral symptoms commonly occuring are independent of cerebral inflammation. Meningitis from the typhoid bacillus has been recorded as occurring without intestinal lesions. Double *optic neuritis* is sometimes seen, but it is rare. Other local inflammations occasionally occur either during the fever or during convalescence, and may considerably delay recovery, such as *parotitis* (·88), which may be followed by suppuration, or extensive infiltration of the neck; *orchitis; periostitis* (1·66), especially of the tibia, or *perichondritis* of the rib cartilages; *myositis; cancrum oris;* abscesses (2·33), boils

(2·2), and facial *erysipelas*. In severe cases *bed-sores* may form, in spite of careful nursing. *Thrombosis* (3·3) of the femoral vein, generally on the left side, may occur during early convalescence, leading to œdema of the foot and leg, and tenderness in the course of the vein. It mostly subsides without much trouble, but the thrombosis may extend into the large abdominal veins, or portions of clot may be detached, and lead to pulmonary embolism and death. *Rigors* are of rare occurrence. They may be due to complications, such as constipation or pneumonia; but they sometimes happen without recognisable cause. Among the nervous sequelæ, besides meningitis, are *encephalitis* (rarely); *mental disturbances* (·44); *peripheral neuritis* (·66), a condition described as *tender toes*, in which the toes and soles of the feet are painful on pressure during attempts at walking; and pain in the lumbo-sacral region aggravated by walking, and persisting for a long time, which has been called *typhoid spine*.

Varieties of Enteric Fever.—There are few diseases more variable than enteric fever. Though its duration is characteristically three weeks, it may be as short as ten days or as long as five or six weeks; and though short attacks may sometimes be fairly represented as abortive attacks, they may be followed by a relapse of precisely the same nature and duration. Sometimes the temperature begins to fall in the manner described (p. 45), and then, before reaching the normal, persists in its remittent type, oscillating between 100° (morning) and 102° (evening) for eight or ten days, so that the fever is prolonged into the fifth week, although the patient is feeling better every day, and has no obvious complications. In other cases the prolongation of the fever corresponds with a continuance of the high temperature characteristic of the second week, and these are generally severe cases. In some cases the illness is so slight that patients go about their ordinary occupations, until, perhaps, an indiscretion in diet, or the use of aperients, given in ignorance, leads to a fatal perforation. Cases so mild as this in their general symptoms, and yet so dangerous from their possible termination, have been called *ambulatory typhoid*. Ataxic and adynamic forms have been described, but these terms simply indicate the predominance of symptoms in one or other system of the body. Very rarely a *hæmorrhagic* form occurs, in which there are purpuric eruptions on the skin, bleeding from the mucous membranes, epistaxis, hæmoptysis, hæmatemesis, and hæmorrhage into the muscles and internal organs. (Compare Measles and Small-pox.) Enteric fever is very often mild in children, often of short duration, and associated with less extensive disease of Peyer's patches than in the average of adult cases. The remissions of temperature, which are well marked in the latter half of the illness in adults, are often still more marked in children, and the "infantile remittent fever" of older writers was undoubtedly enteric fever. In elderly persons, also, the rose spots and enlarged spleen are often absent.

Diagnosis.—A great number of diseases may be confounded with enteric fever, from the variety of forms which it assumes, and from the frequency with which its own typical symptoms are absent or badly marked. In early stages it is distinguished from the exanthems by the absence of characteristic eruption. By the fifth day of the illness, the rash of typhus, small-pox, or scarlet fever would have developed; the appearance of rose spots a few days later confirms the diagnosis of enteric fever. Severe joint pains may lead to a suspicion of rheumatic fever. A prolonged febrile complaint, which has come on insidiously, and presents no obvious local lesions, should always make one think of enteric fever; but the great prevalence of *influenza* gives rise to frequent mistakes. For though influenza is often a much more sudden and quickly prostrating disease, it presents so much variety that almost any illness beginning with headache, backache, and fever is liable to be mistaken for it. If typhoid fever is present the temperature remains high, or even rises, and the diagnosis may be soon confirmed by diarrhœa, enlargement of the spleen, or rose spots.

Later stages present a resemblance to different diseases, according as the head, chest, or abdomen shows the most prominent disturbance. Thus, the early headache of typhoid, and the subsequent delirium, may suggest *meningitis*, and the two diseases are frequently confounded together. Sometimes it is impossible to distinguish them until later stages, when optic neuritis or a local paralysis, squinting or convulsion, or the obstinately retracted abdomen, may decide for tubercular meningitis; or, on the other hand, the increase of abdominal symptoms, with the presence of spots, may prove it to be enteric fever. In this latter, headache rarely continues beyond the tenth day. When pulmonary symptoms are marked, *acute general tuberculosis* may be simulated by the abundant bronchitic râles and crepitations, accompanied by a remitting fever. The abdominal diseases which may be confounded with typhoid fever are, especially, *tubercular peritonitis* and *appendicitis*. In both there may be high fever, abdominal distension and tenderness; and in tubercular peritonitis the stools may be frequent and yellow from accompanying tubercular ulceration. Appendicitis is generally distinguished from typhoid fever by localised pain, rigor, and vomiting, neither of which occurs as a rule in typhoid; but, on the one hand, they are sometimes present in typhoid, and conversely an appendicitis may develop without causing these familiar evidences of acute local inflammation. The *pyæmic* or *septicæmic* condition associated with abscess or suppuration in other parts of the abdomen may also give rise to confusion, such as hepatic abscess and perinephritis: and the rare disease *suppurative pylephlebitis*, in which local evidence of the liver being involved may be little or none, must not be forgotten. In most of these conditions *leucocytosis* is present. An allied condition, *infective endocarditis*, is not infrequently mistaken for typhoid fever. The symptoms in favour of endocarditis are the existence of a murmur, or of irregular action

of the heart, hæmorrhages under the skin, or the evidences of emboli, such as obliteration of the pulse at the wrist or ankle, abundant albuminuria, or retinal hæmorrhages; rigors may be present, and the temperature often oscillates freely. Trichinosis, the disease caused by the multiplication of the *trichina spiralis* within the body, has been mistaken for typhoid fever; it is distinguished by severe muscular pains, œdema of the eyelids, and sometimes of the whole body; and one finds neither rose spots nor enlargement of the spleen.

In most cases of enteric fever the urine contains a substance which gives a reaction—the *Diazo Reaction*—with *Ehrlich's Test*. Though the reaction is nearly always obtained in enteric fever, it occurs frequently in miliary tuberculosis and in measles, and also in some other febrile affections. Its diagnostic value is therefore somewhat limited, and perhaps its absence speaks more against, than its presence does in favour of, enteric fever. In the course of the second or third week the substance disappears and no reaction is obtained: but the recurrence of the reaction after the temperature has fallen indicates a relapse. The test solution, which should be mixed immediately before being used, consists of a concentrated aqueous solution of sulphanilic acid, 200 c.c.; pure nitric acid, 10 c.c.; and a half per cent. solution of pure nitrite of sodium, 6 c.c. Make the urine strongly alkaline with ammonia; then add an equal volume of the test solution. The mixture assumes a red colour; and after standing twelve or twenty-four hours it deposits a sediment, the upper stratum of which shows a light or dark green or blackish-violet colour.

In every doubtful case the *agglutinative* reaction of the patient's serum towards typhoid bacilli, or *Widal's test*, should be examined (*see* p. 19). The test must be carried out by a practical bacteriologist, but the practitioner may collect the required blood from the lobe of the ear in the bulbous portion of a capillary pipette; and sealing the ends of the tube may send it to the laboratory. The test is rarely successful with other diseases than enteric fever; but it is not generally obtained before the seventh day, and unaccountably fails in some instances throughout; on the other hand, the reaction may be shown for months after the termination of the disease. It is most conclusive of typhoid, if the serum diluted to 1 in 30 causes complete clumping in half an hour.

An examination of the blood (*see* Diseases of the Blood) may give some help in diagnosis. In all but the earliest stages there is a reduction of the neutrophile leucocytes, which reach their minimum in the period of declining pyrexia. The lymphocytes are also diminished at first, but increase again at the end of the stage of continuous pyrexia, and remain abundant throughout the fever, and for some weeks into convalescence. Eosinophiles disappear at first, and reappear with the increase of the lymphocytes (Nägeli). Secondary infections, or other complications, may increase the leucocytes again, especially the polymorphonuclear cells.

Prognosis.—The mortality of enteric fever varies in different epidemics from 5 to 20 per cent. Complications contribute largely to the deaths, and their occurrence will modify the prognosis at any time. Apart from them, the intensity of the fever is an important guide. If the temperature is, although high at the end of the first week, subsequently never above 103°, the case is favourable; if the temperature is maintained at 104° or higher throughout the second week, it is much more dangerous. Some cases sink rapidly by the twelfth, eleventh, and tenth days, or even before this. Nägeli finds that the presence of eosinophiles indicates a mild disease, but the absence of lymphocyte increase is unfavourable. Perforation is almost certainly fatal, unless it is promptly treated by surgical methods. Hæmorrhage is less dangerous, but may be responsible for about one-fifth of the deaths; and a severe hæmorrhage, even if not fatal, renders the patient very anæmic, and considerably prolongs convalescence. Much abdominal distension, profuse diarrhœa, severe general bronchitis, and a feeble and irregular heart are all unfavourable.

Treatment.—The patient should be in bed in a well-ventilated apartment, and the same rules should be carried out as to nursing as in the case of typhus fever. The special dangers of perforation and hæmorrhage from the ulcerated bowel should never be lost sight of. Rest should be absolute; the patient should be allowed no exertion, and a bed-pan should be used when he wishes to pass his motions or urine. The diet should consist chiefly of milk, of which two, three, or four pints may be given daily, in regular quantities, every one or two hours, or more frequently. Beef-tea may also be given, but it is said sometimes to increase the diarrhœa, and is certainly not as nutritious as milk. When milk is taken badly, or if the stools show, by the presence of curds, that it is imperfectly digested, it may be peptonised by the use of Benger's liquor pancreaticus. To some, this formation of curds is regarded as a serious objection to milk, as well as the fact that the bacillus typhosus grows readily in milk: whey has accordingly been given with success, although the solid constituents are scarcely more than half those of milk, and the albuminoids are only one-fourth. According to others, a purely milk diet is insufficient, and is responsible for a long and tedious convalescence: these physicians have added farinaceous food, such as arrowroot and corn-flour, as well as beaten-up eggs, and claim that their results are good.

As to medicinal treatment, in mild cases little or none is wanted. A small dose of dilute mineral acid, or of a saline diaphoretic like the acetate of ammonium, may be grateful to the patient; and the body may frequently be sponged with tepid water. With regard to the administration of stimulants, the remarks made in the chapter on typhus apply in the case of enteric fever.

Special symptoms of complications may have to be met, such as bronchitis, by small doses of expectorants; or persistent headache, by phenacetin, 5 to 10 grains. If the bowels are not opened more

than four times in the twenty-four hours, no treatment is required; but it is generally desirable to check diarrhœa if it exceeds this limit, and this is best done by the use of a starch enema with 15 or 20 minims of tincture of opium; and bismuth carbonate or salicylate, or the vegetable astringents, may be given internally. Any linen that is soiled by fæces or urine should be at once removed, not only for the sake of keeping the patient clean and free from the risk of bed-sores, but also to prevent the possibility of the attendants being affected.

If constipation occurs, the bowels may be left for two or three days without harm, and it is then safest to use a soap enema from time to time as required. If there is constipation at the beginning of the illness, in the first week, and before ulceration has begun, a small dose of castor oil may be given. Under no circumstances should the more active or drastic purgatives be employed.

This purely expectant line of treatment suffices in many cases, especially those of milder type: the patient is cared for while the disease runs its course. The methods of treatment by which it has been sought to influence directly the progress and prevent the accidents of the fever are (1) the antipyretic, (2) the antiseptic, and (3) the serum treatment.

The theory of the *antipyretic* method is that the continued high temperature to which the tissues and organs of the body are subjected is the chief cause of their granular degeneration; and that their ultimate failure, or the occurrence of complications, is so much more probable, the higher the fever. Hence, our object should be to bring down the *mean* temperature, by lowering the temperature 3° or 4°, at more or less regular intervals. This theory can scarcely be maintained now, with our modern knowledge of bacteria and toxins. Nevertheless, the practice is sometimes of value in typhoid fever, though it does not cut short the illness.

The methods employed have been already described under the general treatment of fevers (p. 29). Of these the external application of cold gives the best results. The influence of a single bath upon the immediate condition of the patient is generally pronounced. Headache, delirium, stupor, thirst, are at once diminished, the tongue becomes clean, the pulse slower and firmer, and the patient feels altogether relieved. But this effect is only temporary, and by the next observation the temperature may be as high as before. When the system is thoroughly carried out by frequent baths at low temperatures, or even by continuous immersion, the mortality has been markedly reduced, and even in its modified forms a decided improvement has been noted. Its effect upon complications is also marked—bronchitis and hypostatic congestion improve, and it has been shown by statistics that hæmorrhage is less frequent. The contra-indications are extreme collapse of the patient, hæmorrhage already established, and severe conditions of pulmonary congestion. These are likely to be avoided by the adoption of the method from the very commencement of the ill-

ness. One of the great objections is the trouble it entails, especially in private cases; and another is the dislike of the operation which some patients can never overcome: but in some series of cases it appears to have reduced the mortality by 5 or 6 per cent.

Cold sponging, wet packs, the application of ice-bags or ice-compresses to the surface of the body, and the ice-cradle (the patient lying under a bed-cradle within which are hung small vessels containing ice) are less efficient than baths, but they give less trouble, and are often of real service.

Antipyretic drugs have been fully tried, but the disadvantages in the collapse and cardiac failure are generally felt to outweigh their apparent advantages. Antifebrin and phenacetin are least likely to do harm.

The *antiseptic* treatment consists in the use, internally or by enema, of such drugs as carbolic acid, sulphurous acid, naphthol, hydronaphthol, naphthaline, bismuth salicylate, and salol. They are said to diminish diarrhœa and tympanites, and to make the stools less offensive; but they have little or no influence on the changes in the bowel, or on the duration of the pyrexia; nor do they prevent relapse. The doses employed for adults have been of β-naphthol 3 to 5 grains suspended in mucilage every four hours, of hydronaphthol 2 to 3 grains every two to four hours, of sulphurous acid 20 to 30 minims, and of salol 5 to 7 grains.

The following chlorine mixture has also been largely used. Thirty grains of potassium chlorate are mixed with 60 minims of strong hydrochloric acid in a 12-oz. bottle, which is then corked and shaken until it is filled with the chlorine gas. Water is then introduced in small quantities at a time until all the gas is absorbed. Quinine to the extent of 2 or 3 grains per ounce may be added, and one ounce of orange syrup. The dose is one ounce three or four times a day. Dr. Caiger in 1904 spoke well of the oil of cinnamon given in doses of 3 to 5 minims every two hours from the first until the end of the pyrexia.

The *serum-treatment* of enteric fever is yet in its infancy. The recognition that the poison of the typhoid bacillus was mainly an endotoxin, and that little exotoxin was formed, suggested that the bacterial cell juices should be injected into the horse, in order to produce an *anti-endotoxic* serum. Such a serum, employed in the treatment of typhoid fever, has awakened hopes that it may influence the course of the disease (Macfadyen, Hewlett).

For hæmorrhage from the bowels opium internally or morphia by hypodermic injection is probably the best treatment. Acetate of lead, tannic acid, oil of turpentine (10 minims), ergot, and adrenalin chloride have at different times been used; and more recently chloride of calcium in 10-grain doses every three or four hours.

Tympanites may be relieved by the application of ice, in small lumps, between two pieces of flannel, and by the use of turpentine enemata.

If perforation occurs, and is recognised, a laparotomy should be

performed at once, the abdomen should be washed out and the ulcer closed. In default of this, opium in full doses, absolute rest, the application of ice to the abdomen, and feeding by the rectum, must be tried. (*See* Peritonitis.)

Both for the treatment of the *bacilluria* and *cystitis* (*see* p. 49), and for the prevention of infection in others, urotropine (10 grains three times a day), or helmitol should be used during the fever and for three weeks of convalescence.

During convalescence the patient must be kept for at least ten days on fluid food, except in very mild cases, when this rule may be somewhat relaxed. Purgatives must be carefully avoided, or used only in the form of enema. Even in favourable cases without complications or sequelæ, the bodily and mental vigour returns with remarkable slowness, and the patient should not be too early allowed to exert himself. Rarely is one fit for work under three months from the commencement of the illness, and in the graver forms, or in case of relapse, or of complications, this period may be prolonged to five or six months.

Prevention.—The reduction of risks in the supply of water and food forms a large part of the duties of a medical officer of health. But it has been found possible to diminish the susceptibility of the individual to enteric fever by the inoculation of a "vaccine" consisting of dead cultures of typhoid bacilli (*see* p. 21); and persons going on foreign or colonial service, where they are likely to meet with typhoid, are now frequently inoculated. This preventive inoculation is systematically carried out in the Army. A vaccine containing 500 millions of bacilli is first injected, and a second injection of 1000 millions is made ten days later. A certain amount of local and general reaction takes place, but soon subsides. It is estimated that the liability to the disease has been thereby reduced to one-eighth, and the mortality among those who have the disease to one-half.

The treatment of typhoid-carriers presents many difficulties. Intestinal antiseptics are valueless. The use of vaccines has been tried in doses of several millions of sterilised typhoid bacilli at intervals of two or three weeks; but even if the organisms disappear for some time from the urine and fæces the permanence of the cure cannot be guaranteed. The only efficient means of meeting the difficulty of the typhoid-carrier is to keep him under observation and ensure disinfection of his excreta, until he is shown to be permanently free. In purely urinary cases urotropine will reduce the number of bacilli as long as it is being taken.

PARATYPHOID FEVER

It has been found that in a certain number of cases indistinguishable clinically from typhoid fever, the agglutination test fails with typhoid bacilli, but succeeds with bacilli which have a close resemblance to, and yet differ in certain points from, Eberth's bacillus, and belong, indeed, to the Gaertner group. This bacillus

has been called bacillus paratyphosus, and the disease in which it occurs paratyphoid fever. Two types of this bacillus are recognised, differing slightly in their cultural characters: they are called *Bacillus paratyphosus A*, and *B. paratyphosus B.*

It is unnecessary to attempt a detailed account of the symptoms of this form, because they are identical with those of typhoid fever; though from the cases that have been recorded and published, it appears that on the whole the disease is less often severe; that the fever is more often of short duration, and of irregular type, and the mortality is less. Still, on the one hand, the disease is sometimes fatal, and perforation and other serious complications occur; and, on the other hand, as is well known, mild cases of typhoid frequently occur. So that it must not be assumed that paratyphoid fever is simply a mild phase of typhoid fever.

The practical point of course is that in applying Widal's test to a case which resembles typhoid fever, the blood serum must be tested with paratyphoid bacilli of different strains, if it fails to agglutinate typhoid bacilli.

Of the few cases examined *post-mortem*, in some no intestinal lesions were found; in others ulceration of the ileum, not involving Peyer's patches; in one, enlargement of the mesenteric glands; in several, enlargement of the spleen.

MEDITERRANEAN FEVER

(*Malta Fever*)

This is a continued fever of long duration, bearing some resemblance to enteric fever, but distinguished from it by the absence of rose spots, the fact that Peyer's patches are not enlarged or ulcerated, and the low mortality.

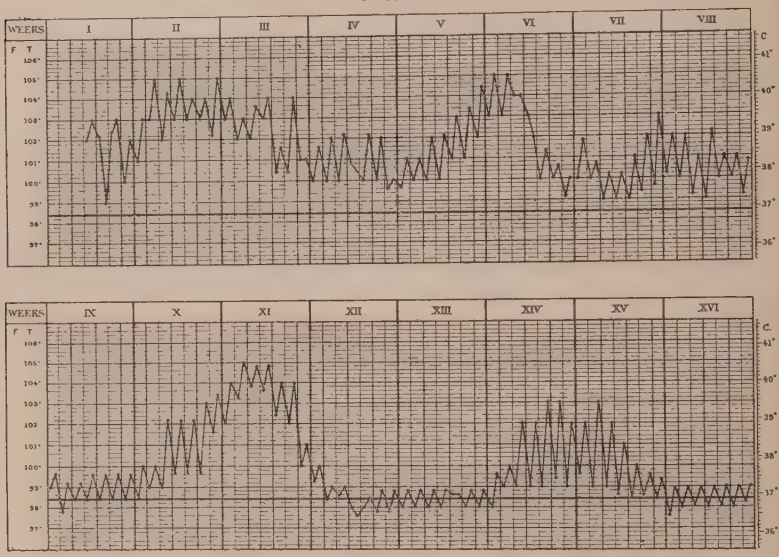
The specific organism is a micrococcus (*M. melitensis*, Bruce), which can be obtained from the blood, from the urine, and by puncture from the spleen during life, and can be found after death in the spleen, liver, kidneys, bone-marrow, and mesenteric glands.

Ætiology.—The disease is prevalent in Malta, and occurs in the other islands and along the coasts of the Mediterranean Sea; and since its bacteriology has been studied it has been proved to exist in India, China, South Africa, the West Indies, and America. It affects the sexes about equally, but is more frequent in the young than in the old, especially between the ages of eleven and thirty. It is more frequent in the warmer months of the year. It has been conclusively shown that the goats, from which the milk-supply is drawn in Malta and other places concerned, are infected with the micrococcus, and that the use of goat's milk as food is the most common cause of the spread of the disease. It may, however, be transmitted by contact of the milk with the abraded skin, and possibly by direct infection, or by means of blood-sucking insects.

Symptoms.—The *incubation* period varies from a few days to three or four weeks. The symptoms come on insidiously, and

consist of pyrexia, headache, pains in the bones, sleeplessness, thirst, furred tongue, loss of appetite, nausea, and weight and tenderness in the epigastric region. The bowels are usually constipated; the spleen is always enlarged, and often tender or painful; sometimes the liver is enlarged; and profuse perspira-

FIG. 6



Temperature in a Case of Malta Fever of Undulant Type.

tions occur with crops of sudamina. The headache and severer symptoms may subside in two or three weeks, but the pyrexia continues for much longer, even up to three, six, or nine months, and only slowly subsides. The temperature is not continuously high over all this time, but has exacerbations of two or three weeks, with intervals of a much lower or nearly normal temperature, so that a close resemblance to the chart of a relapsing enteric fever is presented.

Anæmia is generally marked, and the red corpuscles may fall below three million, while there is a relative increase in the lymphocytes and hyaline leucocytes. In about half the cases the joints become red, swollen, and painful, and the micrococcus has been isolated from the effused fluid. Neuritis is also a common event, especially of the sciatic nerve, beginning acutely and persisting for a long time in a less intense form. In some cases orchitis or epididymitis occurs. The urine is scanty, with uratic deposit and perhaps albumin in acute stages, but generally normal at other times.

The majority of the cases conform to the above or *undulant* type. Exceptionally there are more acute or malignant cases, in which the temperature rises to 106°, 107°, or 108°, and death may take

place from the twentieth to the thirtieth day. On the other hand, there are cases in which the patient is not ill enough to take to his bed. The mortality is only about 2 per cent.

Morbid Anatomy.—The spleen is large, soft, and diffluent, with swollen and indistinct Malpighian bodies, and a great increase of lymphoid tissue; the liver and kidneys are congested, and the latter may be in a condition of glomerular nephritis. The mesenteric glands are slightly enlarged. Peyer's patches are to the naked eye normal; the most that can be seen with the microscope is some slight proliferation of cellular elements.

Diagnosis.—The diseases for which Malta fever may be mistaken are enteric fever, malaria, rheumatism, tuberculosis, and septicæmia. The diagnosis can generally be made by the agglutination or Widal's test: the serum of the patient agglutinates the micrococci sometimes as early as the fifth day in dilutions of 1 to 100 or 1 to 1000, and this action persists far into convalescence, even for years. The micrococcus can also be isolated from the peripheral blood, by puncture from the spleen, and from the fæces (Eyre).

Treatment.—Neither quinine nor sodium salicylate has any influence on this disease. It may be treated in the same way as a mild enteric fever by milk diet; and troublesome symptoms must be met as they arise. For the joint affection the use of iodine, or friction with liniments, is recommended (Bruce).

Prevention.—This requires notification of cases of the disease to the proper authorities, frequent testing of the milk supplied by the goats, and segregation of the infected animals. The test employed is the "Zammit test," which depends upon the fact that the specific *agglutinins* are present in the milk, and will clump the micrococcus as in the Widal test for typhoid fever. Sedimentation (*see* p. 19) occurs on mixing equal quantities of a 1 in 10 dilution of the milk with an emulsion of *M. melitensis* in distilled water. Temporary protection appears to be provided to the individual, who is exposed to infection, by inoculation with a vaccine of the micrococcus.

WEIL'S DISEASE

The illness described under this name on the Continent, but rarely seen in England, is an acute febrile disease, of short duration, accompanied with jaundice and swelling and tenderness of the liver. It is most common in young adult males. As a large proportion of the patients have been butchers, and the disease has occurred in the hot season of the year, it is likely that the disease arises from contact with decomposing animal matter. It begins suddenly, often with a chill, and without prodromal symptoms; the symptoms are fever, headache, signs of gastric disturbance, jaundice, and muscular pains, especially in the calves. The fever lasts eight or ten days. Sometimes there is a relapse. The pulse is quick at first, and afterwards slower than normal. The spleen and liver are commonly, but not always, swollen, and

the liver is often tender on pressure. Nephritis is often, and herpes and erythema are occasionally, observed. The recorded cases are at present not numerous, deaths are comparatively rare, and the nature of the disease is uncertain. It is not any form of enteric fever, nor relapsing fever, nor catarrhal jaundice. In some of the fatal cases, an organism—the *Bacillus proteus fluorescens*—has been obtained from the urine and pus during life, and after death from the kidneys, lungs, spleen, liver, bile, and blood from the heart.

SCARLET FEVER

(*Scarlatina*)

Scarlet fever is a contagious disease, characterised by fever, sore throat, a bright red eruption on the skin, and a tendency to certain complications, of which the most important is acute inflammation of the kidneys.

Ætiology.—Scarlatina is usually derived from a preceding case of the disease, either by direct contagion from the breath or exhalations of the sufferer, or by minute particles of shed epithelium, or by dried secretions from mucous surfaces. It is much more readily conveyed to a distance than typhus or relapsing fever, and it is remarkable for the tenacity with which it adheres to clothing, bedclothes, books, papers, and other articles that have been used by the sick; so that the contagium has been conveyed by such means over miles of country, or has lain dormant for weeks or months, and then, meeting with a suitable nidus, has again developed the disease in its complete form. The infection is also conveyed by milk used as food, and though the milk has sometimes been infected by contact with scarlatina in the person of the milkman or his associates, it is very probable that it may be infected from a diseased condition of the teats or udder of the cow supplying it. Sex, occupation, and social position have no influence in its production, but the vast majority of those attacked by scarlatina are young children; it is comparatively infrequent in adults, and very young infants are less susceptible than older children. The relative exemption of grown-up persons is explained by the fact that, for the most part, one attack protects against future attacks, and that many adults have already had the disease. Still, old people occasionally have it; and protection is not always perfect, so that some people have a second attack.

Symptoms and Course.—The period of *incubation* is two, three, or four days, rarely as much as six days. Generally the invasion is sudden: the patient has a rigor, or vomits, and complains of frontal headache, with languor, pains in the back and limbs, and loss of appetite. The temperature rises to 103° or 104°, the pulse becomes very rapid, and the respiration is quickened. Very soon there is some complaint of sore throat, and swallowing is painful.

On the second day—that is, generally between twelve and

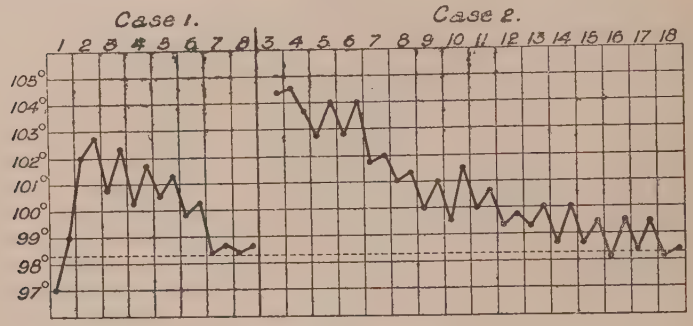
thirty-six hours from the first symptom—the *rash* appears. It is first seen on the upper part of the chest, in front and on the sides of the neck, but soon spreads to the abdomen and back, and then to the upper and lower limbs. It consists of minute red spots, bright in the centre, fading towards the edge, and set closely together, so that the paler edges almost coalesce. Sometimes the coalescence is complete, so that the skin has a uniform bright red colour; sometimes the eruption is more discrete, and areas of pale skin are visible between the spots. The face, forehead, and cheeks are as a rule deeply and uniformly flushed, without showing the punctiform arrangement of the rash which is seen elsewhere; but the skin round the mouth remains pale. With an abundant rash the skin becomes slightly swollen. The eruption presents many varieties as to depth of colour and distribution. It may be only pale pink, or it is deep, livid purple; and in some severe cases papules may be raised above the surface, and may even vesicate or form minute points of pus; and occasionally petechiæ occur. In its distribution the rash may be very limited, occurring only on the chest, or in patches on the thighs, elbows, or ankles, and this occurs frequently in second attacks, and in the mild cases sometimes seen in patients with open wounds, formerly known as "surgical scarlatina." The rash reaches its height on the third or fourth day, and begins to fade on the fourth, fifth, or sixth day; altogether it may last from five to ten days. After the subsidence of the rash, *desquamation* takes place—that is, the superficial layers of the cutis are shed. This occurs in the form of white, branny flakes on the sides of the neck, preceded (as pointed out by Caiger) by an appearance of pin-point depressions, due to the rupture of the epidermis at the top of each papule. This may be as early as the sixth or seventh day, while the eruption is still visible on the legs; but the amount of epithelium that is shed, and the size of the particles, are very variable—sometimes there is nothing more than a little roughness about the tips of the fingers or toes, or in the folds of the palms of the hands; while in other cases the epidermis peels off in large flakes, and, in rare instances, complete glove-like moulds of the hands and fingers are thrown off. Desquamation commonly takes from four to six weeks, but in these special cases a much longer time is required.

In the throat it is seen that the uvula, soft palate, and fauces are deep red, and often slightly œdematous; the tonsils are reddened, swollen, projecting towards the middle line, and presenting a number of yellow points, from the follicles being distended or covered irregularly with ashy or yellowish secretion. In later stages they may suppurate, or sloughs may form in them. The nasal mucous membrane also inflames, and secretes a quantity of mucus; and the submaxillary and cervical glands become enlarged and tender. The tongue is at first thickly covered with white fur, but in a few days this clears off from tip to base, leaving a bright red, raw surface, on which the fungiform papillæ are unusually

prominent, so as to give the appearance known as "strawberry tongue."

The temperature is frequently high, reaching 104° or 105° on the first day, and remaining at this level for some days. Even a temperature of 106° may be reached. With this the skin is pun-

Fig. 7



Temperature in Scarlatina.

gently hot and generally dry, but profuse sweating may occur without appreciably affecting the temperature or the rash. The pulse rises to 120, 140, or even 160. In severe cases, the mental faculties are dulled; delirium is frequent, especially towards night; and drowsiness and coma supervene. The disease may reach its height about the fourth, fifth, or sixth day, and then, with the fading of the rash, the temperature begins to fall, generally subsiding rather gradually, but sometimes more suddenly, till the normal is reached, and convalescence is gradually established. In fatal cases death may occur about the fifth day or later from exhaustion; or from typhoid conditions, with low delirium, semi-coma, and dry brown tongue; or it may occur later still as a result of complications.

Complications and Sequelæ.—These are numerous and important. Not only the tonsils, but also the soft palate and the uvula, may slough. More frequently the *glands* under the jaw and in the neck are much swollen, and the subcutaneous tissue about them is infiltrated, becoming brawny and indurated. The skin then becomes dusky red, and sloughing takes place beneath it, separating it from the subjacent tissues over a large area. Such cases are often fatal. Extension of the inflammation from the throat up the Eustachian tube may cause *otitis*, or inflammation of the ear, resulting in abscess of the tympanum, rupture of the membrana tympani and otorrhœa. In the course of the scarlatina this may seem of little importance; but it lays the foundation for serious or fatal results months and even years afterwards, among which may be enumerated suppuration of the mastoid cells, meningitis, abscess of the brain, thrombosis of the lateral sinus or jugular vein, with pyæmia as a result, hæmor-

rhage from the lateral sinus, and facial paralysis. Deafness on the affected side may of course happen; and a double otitis in a young child may be the cause of permanent deaf-mutism. Other local lesions may occur as sequelæ; for instance, sloughing of the cornea, abscesses in the subcutaneous tissues, or cancrum oris.

The kidney is often involved in connection with scarlatina, and nephritis and albuminuria may occur in the following three ways: Albuminuria is occasionally present in the stage of rash and fever; small in quantity, temporary in duration, and probably arising in the same way as it does in severe cases of other infections. Secondly, nephritis may be first recognised as a sequela from two to three or four weeks after the beginning of the illness, when the patient is quite convalescent or suffering only from desquamation. It then begins with a chill and rise of temperature, and the passage of turbid brown or blood-coloured albuminous urine, the whole subsiding again without the occurrence of dropsy. In a third group of cases the first thing noticed is some swelling of the feet and face, and then the urine is found to be scanty, high-coloured, and albuminous, with blood-pigment and granular, hyaline and epithelial casts. Recovery from slight cases is common; but the dropsy may become general, and death may result after six, twelve, or eighteen months, with the severe secondary complications which will be described elsewhere. (*See* Nephritis.)

Pneumonia, broncho-pneumonia, bronchitis, pericarditis, and *endocarditis* occasionally occur in the course of the illness. The first two are responsible for one-eighth of the deaths. Broncho-pneumonia may be caused by inhalation of septic materials from the throat. *Pleurisy* may happen as a sequela; and if effusion take place, it often becomes purulent quite early. As in other severe fevers, dilatation of the *heart* sometimes occurs, and is recognised by displacement of the impulse. An acute general *arthritis,* which is indistinguishable from rheumatic fever, often follows upon scarlet fever so closely that the joints may be swollen when the rash is still present. Although generally known as *scarlatinal rheumatism,* it is possibly a synovitis due to the direct action of the septic organisms of the primary disease. It may affect many joints, but is not, as a rule, severe; and it may be accompanied by endocardial murmur, and followed by permanent valvular disease. It is often valuable as clinching the diagnosis in a doubtful case of scarlatina. Exceptionally, the joints suppurate. Some relations of scarlatina to diphtheria are mentioned under the latter (pp. 120, 124).

Varieties.—Besides the ordinary forms of scarlatina of moderate severity, which end in recovery, one recognises cases that are called *scarlatina maligna.* This form—also called, but not very logically, the *toxic* form—mostly includes cases that are fatal within five or six days from the intensity of the disease, without complication other than sore throat. Sometimes the patient is struck down with convulsions and collapse, and dies in twelve or twenty-four hours, before the rash has had time to develop. In other cases there are

severe rigor and vomiting, early intense or livid rash, high fever and delirium; and the patients die in two or three days.

Cases with severe throat-symptoms have been called *scarlatina anginosa*. To this nearly corresponds the *scarlatina ulcerosa* or *septic form* of Caiger, in which the faucial ulcers form a septic focus, from which the system may be poisoned.

The term *latent scarlatina* includes cases in which the rash and sore throat have been so slight as to escape detection, and the illness has only been discovered by the occurrence of desquamation or anasarca. Patients with open wounds appear to be particularly susceptible to scarlatina, probably because the wound provides an easy entrance for the poison. The disease is generally mild, the rash partial and of short duration: so that the connection with scarlatina was for a long time misunderstood.

Morbid Anatomy.—The organs, after death from scarlatina, present little that is peculiar. In malignant cases there are the changes (*see* p. 26) common to the pyrexial and septic disorders:—undue fluidity of the blood, soft liver and spleen, petechial spots on the serous membranes, and hypostatic congestion or œdema of the bases of the lungs. The tonsils present the conditions of ulceration or suppuration that have been observed during life. Where complications are present, the visceral lesions proper to them will be found.

Further observations point, as formerly, to the likelihood that a streptococcus (*S. Scarlatinæ* or *S. conglomeratus*) is the specific micro-organism of scarlatina: and Schleissner states that by the "complement" test (*see* p. 143) it is found that the serum of scarlatina patients contains a specific anti-body for the scarlatina streptoccus. But the question still remains open. Many of the complications involving the throat, ears, and other parts are secondary affections caused by the usual pyogenic organisms.

Diagnosis.—Scarlatina is recognised, especially when the disease is known to be prevalent at the time, by the occurrence of feverishness with sore throat, followed in a day by the characteristic rash. The rash may be confounded with that of *measles*, *rubella*, or *typhus*, or with the roseola of *small-pox* and other fevers. Generally, it is of brighter colour and more uniform distribution than those of the first three diseases; but it is sometimes very difficult to distinguish from rubella. The roseola of small-pox often has a distribution which is distinctive—namely, about the axilla, groins and thighs; and it is more purple in colour. Occasionally, confirmation only comes with desquamation and the appearance of albumin in the urine.

The **Prognosis** must be in all cases very uncertain. Even in the mildest cases, renal complications may be serious or fatal. The mortality, however, is variable, some epidemics being exceedingly mild, others so severe that the mortality may rise to 30 or 40 per cent. In individual cases, the prognosis may have to be determined by the condition of the patient from day to day; the addition of complications increases the danger. Very severe angina, and an intense or livid rash coming out late, are unfavourable; and cases

with sloughing of the cervical glands are generally fatal. Dr. W. Hunter states that both the initial angina, the adenitis, and the complications in the throat, nose and ears are more severe in those previously suffering from bad teeth, inflamed gums, or pyorrhœa alveolaris (oral sepsis), than in others whose mouths are perfectly healthy. Scarlet fever attacking women recently confined shows a large percentage of fatal cases.

Treatment.—A specific treatment of scarlet fever has only been possible in recent years. Antitoxic sera prepared from streptococci cultivated from scarlatina cases have been used rather frequently abroad, and are said to modify the course of the disease favourably. They are still on their trial.

Apart from this, the treatment must be carried out in the same way as that of other fevers: a well-ventilated room, the recumbent position in bed, light diet, and careful nursing. Isolation is essential in the interests of others. The body may be usefully sponged with tepid water daily; simple salines may be given internally, and the sore throat may be relieved by sucking lumps of ice. If the tonsils are much swollen, and much covered with secretion, the latter should be removed by pledgets of moist lint, and disinfectant or astringent solutions applied by the same means. Formalin (1 in 200), liquor ferri perchloridi (℥ss to aq. ℥j), dilute hydrochloric acid (℥ss to ℥j), or liquor sodæ chlorinatæ or izal may be thus used; and similar remedies, more diluted, may be used to syringe the nose when that is involved. Irrigation of the fauces every four hours with about two pints of warm water delivered from a reservoir two feet above the patient's head, which may be turned over the side of the bed, is desirable to prevent absorption of toxins from the throat (A. K. Gordon). If a condition of oral sepsis is present, it should be treated with antiseptics from the first. For pain or swelling in the neck and about the angles of the jaws, hot fomentations or boric lint wrung out of hot water should be used. Complications will require special treatment. Abscesses should be opened early. In otitis, relief of pain may be obtained by hot fomentations, and the gentle introduction of warm water into the meatus. If suppuration of the middle ear is recognised, the membrana tympani may require puncture; and the meatus may be gently syringed with warm water or diluted Condy's fluid, or solution of boric acid (1 in 20). For synovitis salicylate of sodium should be given in ten or fifteen-grain doses, and chloroform or belladonna liniment may be used locally. The treatment of scarlatinal nephritis will be considered hereafter (*see* Nephritis). In the severe typhoid forms, with quick, feeble pulse, stimulants, such as brandy and ammonia, must be given; and in these toxic cases Gordon recommends a polyvalent antistreptococcic serum in doses of at least 50 c.c. Where there is very high fever, with much delirium and restlessness, relief is often obtained by cold affusion to the head or body.

Great care must be taken during convalescence, both for the sake of the patient and in the interests of the public. The

occurrence of nephritis is probably favoured by, though not entirely dependent upon, exposure to cold; this should, therefore, be especially guarded against as long as desquamation is going on. A free action of the bowels should also be maintained by occasional saline laxatives. During free desquamation, the body should be washed night and morning with soap and warm water, and gentle friction with pumice-stone may be employed to remove the desquamating epithelium; and in the intervals the body should be smeared with carbolic oil (1 to 40) or with glycerin to prevent the particles being carried off into the atmosphere. Quinine and other tonics may be necessary where strength is recovered slowly.

The frequency with which convalescent patients returning from fever hospitals to their own homes have conveyed the disease to their brothers or sisters, not till then infected, has given rise to the name "return cases" for the new sufferers. There is much evidence that in these cases the contagion is far more often transmitted by secretions from the throat, nose, or ear, than by the desquamating skin, and as much attention should be therefore directed to the former as to the latter. Hence, while no patient should be allowed to mix with the unprotected till six weeks have elapsed, it would still be wise to prolong this period in cases of tardy desquamation or persistent discharge from the mucous surfaces.

MEASLES

(*Morbilli*)

Measles is a contagious febrile disease, characterised by an eruption of pink or red spots, and catarrh of the respiratory mucous membranes.

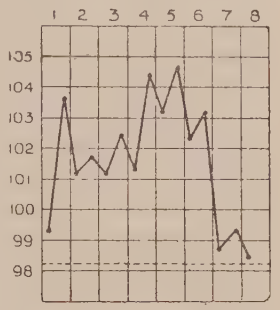
Ætiology.—In civilised communities its spread is determined by circumstances very similar to those influencing scarlatina. It occurs in epidemics, which attack the young rather than the old, chiefly because nearly all the older members of the community have had the disease when young, and are thereby protected from a second infection; but very young infants appear to be less susceptible, and to have the disease, when caught, in a milder form than children somewhat older. In large towns it is almost continuously present, spreading from point to point in the form of limited outbreaks, which subside, and are succeeded by others in different places; but where introduced among populations that have never been visited by the disease, or have been entirely free from it for years, it attacks the majority of the people, young and old alike, in one great and often destructive epidemic. This was the case in the Faröe Islands in 1846.

Infection is mostly produced by contact with the patient, or with the air infected by him; the virus also adheres to clothes and other articles, though with much less tenacity than does the contagion of scarlatina. Experimentally also, the disease has been

transmitted by inoculation with the nasal mucus of a patient. It is especially contagious in the prodromal stage preceding the rash, and in the stage of eruption itself.

Symptoms and Course.—The period of *incubation* varies in measles as in other contagious diseases, but has been most often found to be ten or eleven days. The disease commences with pyrexia and catarrhal symptoms—the temperature rises perhaps to 102°; the child loses appetite, is drowsy, and unwell; there may be, at first, vomiting or chills, or, in children, convulsions. With this the conjunctivæ become suffused, the eyes water, there is a mucous discharge from the nose, and cough as a result of bronchial secretion. The catarrhal symptoms continue, but the temperature frequently falls after the first day, and continues at a lower level for another day or two, when it again rises. Occasionally in this early period there is a so-called prodromal rash, which may be like that of scarlatina, or urticarious, or like the true measles rash. It lasts only twenty-four or thirty-six hours.

FIG. 8



Temperature in Measles.
(*After Strümpell.*)

The characteristic eruption appears most commonly on the fourth day, but it may be as early as the third. It is first seen on the face, at the roots of the hair, on the forehead, temple, or behind the ears, and it subsequently spreads to the neck, trunk, and limbs. It consists of pink spots, round, oval, or irregularly shaped, slightly raised above the surface, running together into irregular groups, which may have a somewhat crescentic shape, and leave some intervening area of skin unaffected. In colour, it is generally darker red, or more purple than that of scarlatina; but a distinction may be difficult, especially if the spots are uniformly distributed and do not coalesce. Occasionally, a few petechiæ occur in the darkest part of the eruption, and in other cases a few vesicles may form in the centres of some papules. It comes out most fully on the face, giving it a blotchy, swollen appearance, and though less abundant on the extremities, it may form continuous patches of infiltration on the back and arms. It takes from one to three days to reach its height, and then rapidly declines, mostly beginning to fade first where it first appeared. It commonly leaves some mottling of brown or yellowish-brown colour, which lasts for some days; while petechiæ leave still more pronounced stains. It is also succeeded by slight desquamation in minute branny scales, and never in the large flakes seen in scarlatina.

If the temperature has fallen in the prodromal stage, it rises again, with the appearance of the rash, to 102°, 103°, or even higher, and reaching a maximum in two, three, or four days, falls generally rather suddenly as the rash begins to fade, and may

reach the normal in about thirty-six hours. The catarrh continues throughout the eruptive stage; it may extend into the frontal sinuses and cause headache. There is more or less general bronchitis, indicated by cough, expectoration of mucus, and diffused rhonchi; and the larynx may be implicated, as shown by hoarseness, croupy cough, and in occasional cases by stridulous breathing. Even before the appearance of the rash, the palate often shows abnormal redness, diffused or in pitches, and almost invariably the lesions known as *Filatow's* or *Koplik's spots*. These are small, raised, white or opal dots, the size of a small pin's head, generally on a reddened base. They are seen best on the buccal mucous membrane opposite the premolar teeth of the lower jaw, and to a less extent opposite the other teeth. These appear two or three days before the eruption. The tongue is usually furred, and the fungiform papillæ are prominent. After the subsidence of the fever the return to normal appetite and sleep is generally rapid.

Complications and Sequelæ.—The most important complications are those connected with the respiratory organs, and it is to them that the majority of the deaths in connection with measles are due. *Pneumonia*, or inflammation of the lungs, is of frequent occurrence, and has generally been attributed to an extension of the bronchitis, which is common in all cases of the disease. But the pneumonia may be lobular, or lobar, in its distribution, and it may present in one case the features of a broncho-pneumonia, and in another case those of a croupous (pneumococcal) pneumonia. The *laryngitis* may be so severe as to threaten asphyxia, and may be accompanied with the formation of membrane; in some such cases it is a true diphtheria; in others it is caused by pyogenic organisms. Other complications are ophthalmia, keratitis, or iritis, stomatitis and parotitis, inflammation of the Eustachian tube, and of the tympanum (*otitis media*), and diarrhœa or dysentery from enteritis of the small or large intestine respectively. Noma, or gangrenous inflammation of the mouth or of the vulva, occurs occasionally. Amongst sequelæ may be mentioned, especially, chronic inflammatory conditions of parts affected during the illness, such as chronic catarrh of the respiratory organs, chronic ophthalmia, enlarged tonsils, or enlarged glands, otitis with discharge and its results (*see* p. 62), tuberculosis, endocarditis, and general malnutrition.

Varieties.—Measles without eruption and measles without catarrh have both been described; but there seems to be some doubt whether the former occurs, and some cases of the latter may really have been instances of rubella or German measles (*see* p. 70). In either case, the disease takes on a mild form. Of the severer or malignant forms, *hæmorrhagic* measles is an example, in which bleeding takes place from mucous membranes, and the eruption becomes *purpuric*. Other severe forms are merely characterised by intense fever, dark or livid rash, often imperfectly developed, rapid and feeble pulse, prostration, delirium, dry brown tongue, and a generally typhoid condition.

Morbid Anatomy.—This must depend much on the complication causing death, since uncomplicated measles is so rarely fatal. Redness of the mucous membrane of the larynx and trachea is observed; the spleen is moderately swollen. No special description is needed of the bronchitis and pneumonia found in cases dying therefrom. There may be, however, fluid in the pleural cavity, and petechiæ under the pleural membrane; the bronchial glands are often enlarged and softened. Congestion of the mucous membrane of the ileum and colon is also sometimes found.

No micro-organism has been identified as the certain cause of measles.

Diagnosis.—A confusion with *scarlatina* is most likely to occur. Measles is distinguished by the initial coryza, by the longer prodromal period, and the character of the eruption, especially in its patchy appearance, as distinguished from the uniform or finely punctate distribution in scarlatina. But the throat may be sore in measles, and the rash sometimes appears on the third day, so as to make the resemblance to scarlatina rather close. A prodromal rash would also increase the difficulty. In measles the blood shows a diminution of the leucocytes, whereas in scarlatina, there is an increase of the polymorphonuclear cells. Measles may also be mistaken for *rubella*, in which the fever is slight or none, the prodromal period short or absent, catarrhal symptoms wanting, the rash in smaller patches, more uniformly distributed without crescentic arrangement, the liability to complications very small, and a fatal termination unknown. In *typhus* the rash is not so papular, the face is but little affected, the spleen is swollen, and there is no nasal or conjunctival catarrh. The early stage of a *small-pox* eruption is sometimes simulated by that of measles; the absence of catarrh, and the history of headache, back-pain and sickness, are in favour of variola. Roseolous eruptions, apart from specific fevers, may resemble measles, but will be distinguished by the absence of the characteristic fever and catarrh. Koplik's spots are of value in diagnosis, as they rarely occur in other eruptive diseases.

Prognosis.—This is usually favourable. For the most part, the mortality is from 1 to 2 per cent., though occasionally epidemics of much greater severity have occurred; and the prevalence of pulmonary and laryngeal complications increases considerably the percentage of deaths. Apart from these, the malignant cases are recognised by intense fever, dark or livid eruptions, and early collapse or prostration.

Treatment.—The treatment of measles is not essentially different from that of scarlatina. The child must be placed in a suitable room—warm, well ventilated, and free from draughts, and so arranged as to prevent infection of children hitherto free. Confinement to bed is scarcely necessary till the eruption appears. The diet, as in other febrile affections, must be mainly milk, with a little farinaceous food. The catarrh which is present from the

first should be treated by expectorants, such as squills, or ipecacuanha wine, or small doses of compound tincture of camphor. The application of glycerin, or borax and glycerin, to the fauces and throat will relieve the harder kinds of cough. If fever runs high, it may be reduced by the application of tepid water, either by sponging or by immersion in a bath at 95° or 90°, which may be lowered still more by pouring in cold water. The temperature seldom remains at a high level, such as 105°, for many hours, but it may be desirable to spare the patient this prejudicial condition as much as possible; and even at a temperature of 102° or 103° a good deal of comfort and even sleep may be obtained by the use of cold sponging, as has been shown when treating of enteric fever. Stimulants are only required in the very severe forms. The child may generally be allowed to get up two or three days after the subsidence of the fever, but should be confined to the room for another week or ten days. During convalescence the general health should be attended to, and iron or cod-liver oil may be given, with, perhaps, change of air to the seaside or other bracing locality. Pneumonia or diarrhœa may be treated in the usual way, and discharge from the ears should be met by frequent washing with antiseptic lotions—*e.g.*, potassium permanganate or boric acid.

RUBELLA

(*Rubeola, Rötheln, German Measles*)

This is an exanthem, resembling in many points both measles and scarlatina, but undoubtedly distinct from both. No specific micro-organism has yet been discovered in connection with it.

Ætiology.—It is very contagious, exposure to the surroundings of a patient for a few minutes being sometimes sufficient for infection. The conditions of its transmission are similar to those observed in measles: it is perhaps less frequently epidemic, and hence a larger proportion of people escape.

Symptoms.—The period of *incubation* is often sixteen or seventeen days, and may be a few less or more. A prodromal stage is either entirely absent, or at most lasts half a day, before the appearance of the eruption; and this stage may be represented by a slight catarrh of the mucous membranes of the air-passages or of the conjunctiva. But in some cases the *eruption* is the first indication of anything wrong with the patient. It consists of a number of pink spots, round or oval, very slightly raised above the surface, uniformly scattered, and generally discrete, though sometimes very closely set. The spots vary in size; when small and closely set there may be much resemblance to a scarlatinal rash; when larger there is more likeness to measles, but they are not commonly confluent, and do not take any crescentic form. Slight itching of the skin may be experienced. The eruption occupies the face, trunk, arms, and legs, appearing mostly on the face first,

and rapidly occurring on the other parts; it is generally of shorter duration than measles, often lasting only two days, sometimes three or four. As in measles, it may leave a little discoloration of the skin for some days afterwards; desquamation is commonly absent, and it is never in large flakes, as in scarlatina. The palate and fauces usually show some injection or spots and streaks of redness, and the tonsils may be a little swollen. The conjunctivæ are reddened, and coughing and sneezing are generally present to a slight extent. The *lymphatic glands* at the back of the neck are frequently swollen, and sometimes those in other parts of the body. The swelling may persist two or three weeks, but suppuration has never been observed. Fever is, in the majority of cases, entirely absent; if it occurs, the temperature is only 1·5° or 2° above the normal, and it lasts one, two, or at most three days, showing the greatest variability in different cases, but often falling to normal before the eruption is completely developed. Many patients do not feel ill at all, and retain their appetite throughout. Any further complications than those indicated already are quite uncommon, and the prognosis is exceedingly favourable.

Diagnosis.—This has to be made from *measles* and from *scarlatina*. As against the former, note the absence or shortness of the prodromal stage; the slightness of the fever and of the catarrhal symptoms; the round or oval shape, with smaller size and paler colour of the spots, which do not run together into extensive or irregular confluent patches; the rapid extension of the eruption, or its outbreak over the whole body at the same time. From irregular and mild forms of scarlatina it must be distinguished by the larger size of the spots and their more scattered distribution, and by the extension of the eruption on the face up to the lips, contrasting thus with the circumoral pallor of scarlet fever. It is stated that the leucocytes are diminished in number, with a relative increase of the uninuclears (*see* p. 69).

Treatment.—This must be conducted on the lines laid down for measles.

SMALL-POX

(*Variola*)

Small-pox is a specific contagious disease, with a characteristic pustular eruption.

Ætiology.—This disease arises solely by contagion—chiefly, no doubt, by inhalation of the atmosphere surrounding infected persons; but it is also conveyed by clothes, bedding, and other things, which have been in contact with patients; and it can be inoculated by means of the contents of the pustules. But patients are infectious before the eruption, and the virus is given off even from the bodies of those who have died. The susceptibility to the disease is common to all ages and both sexes; even the fœtus *in utero* may catch it from the mother; but the susceptibility then and

in the first year of life is stated to be less than afterwards. Negroes are said to be more liable than white people. The liability to the disease, and consequently the number and severity of its epidemics, have been reduced considerably since the introduction of vaccination at the end of the eighteenth century. The disease commonly occurs only once in the same individual; but second and third attacks occasionally occur, and the second attack may even be more severe than the first, though it is generally milder.

Symptoms and Course.—The period of *incubation* is, in a large proportion of cases, about twelve days, during which, as a rule, the patient is well. The disease mostly begins suddenly with a distinct rigor or chills, with pains in the back—that is, the loins and sacrum—severe headache and vomiting. The temperature rises rapidly to 102°, 103°, or 104°, and the next day it may be still higher. The patient is mostly very ill, is unable to continue his work, and probably takes to his bed. Anorexia, thirst, furred tongue, and constipation are also present. On the third day the typical eruption appears; but in the initial stage in a certain number of cases rashes occur, with which it is important for diagnostic purposes to be acquainted.

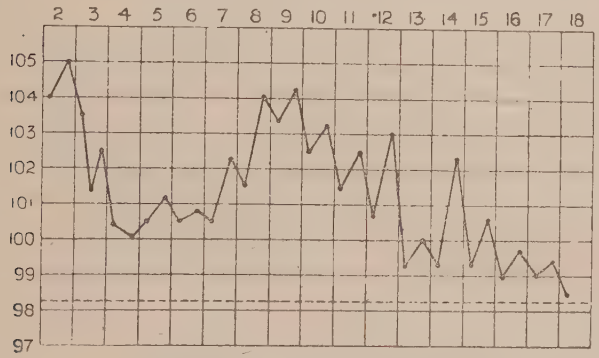
Early Eruptions.—These are either *erythematous* or *hæmorrhagic*. Of the erythematous rashes, some cover the whole body and face, and either closely resemble scarlatina or are more like measles; in other cases the rash is partial in its distribution, and has been especially noted on the external surfaces of the arms and legs. Of the hæmorrhagic rashes the most characteristic is one which occupies the lower half of the abdomen, from the umbilicus downwards, covers the groins, and extends on to the thighs in a triangular form, with the apex downwards, corresponding closely to Scarpa's triangle; it also frequently appears in the axillæ, and on the adjacent parts of the arms and trunk, and extends thence along the flanks to the lower patches. It consists of small hæmorrhagic spots, or petechiæ, which, on fading, leave brown or yellowish-brown stains for a time. These initial rashes commonly appear on the second day, and last for about two days, co-existing, perhaps, with the early stage of the pustular eruption, but disappearing before its full development.

Another form in which an initial rash appears is that of the *purpura variolosa*, which constitutes a very severe variety of the disease. On the second day, or even within twenty-four hours of the first symptom, a scarlatiniform rash appears, quickly followed by subcutaneous hæmorrhage, partly petechial, partly in larger patches. The face is red and puffy, the eyes suffused; there may be vomiting of bilious matters or of blood, with the passage of bloody stools, and the urine contains albumin or blood. The mind is generally clear till near the end; exceptionally, delirium or coma is observed. The cases are nearly always fatal, often within three days of the commencement, and even earlier.

Specific Eruption.—This commonly begins on the third day

of the illness, by the formation of small red papules on the face, forehead, and scalp, and the same appear subsequently on the chest, back, arms, and hands; finally, on the lower part of the body, the legs and feet. These papules soon become prominent; they are firm, and give to the finger the impression of extending

Fig. 9



Temperature in Small-pox. (*After Strümpell.*)

deeply, a condition sometimes described as "shotty." On the third day of their appearance a small vesicle appears in the centre, which is at first clear and transparent. As it gets larger, during the next two days, a very characteristic change takes place: the centre becomes depressed and the circumferential part forms a prominent ring round it. This process is known as the *umbilication* of the vesicle. If the vesicle be punctured only a small quantity of the contained serum will escape, the retention of the remainder being due to septa which divide the vesicle into separate cavities or *loculi*. Almost coincidently with the umbilication of the vesicle, the contents become more opaque; and finally, about the sixth day (eighth of the disease), they are completely purulent. During this change in the vesicle, the surrounding skin becomes pink, forming an inflammatory halo around it, and if there are many pustules—for instance, on the face—this leads to a great deal of swelling, which is often so considerable as to render recognition of the features quite impossible. The scalp becomes tense and tender; and the fingers also are often much swollen from the same cause. The stage of suppuration lasts two or three days, and then the pustules gradually dry up, beginning at the centre, and ultimately forming a brown or blackish-brown scab, which adheres for several days. Sometimes the desiccation is preceded by the escape of some pus from the vesicle, and with the drying up the swelling of the face and other parts subsides. Finally, the scab falls off, leaving a dark red spot, which is at first slightly raised above the general surface, but in the course of some weeks forms a depressed white scar. The pustules form most abundantly on the face, and on

the backs of the hands, and are less numerous on the trunk and covered parts of the limbs. Parts that have been the seat of initial erythematous or petechial rashes are by many said to be less liable to the specific eruption. All the stages of the eruption occur first on the face, and follow, a day or so later, on the trunk and extremities. On the other hand, parts which have been irritated, as, *e.g.*, by the application of plasters or blisters, are liable to an abundant formation of pustules. The pustules are not confined to the skin, but occur on the mucous membranes also; they are especially well seen in the mouth, on the hard and soft palate, but present different appearances from those on the skin on account of the constant moisture to which they are subject. They scarcely develop into well-formed pustules, but are only gray or pearly elevations, which are liable to become abraded and form superficial erosions or ulcers. The tongue is generally coated and more or less covered with pustules; rarely its substance is inflamed. Ulceration of the larynx or even perichondritis may occur; and the process may extend to the nasal mucous membrane, so that the breathing is obstructed by the swelling and the formation of scabs.

General Condition.—In mild cases the *primary fever* falls with the appearance of the specific eruption, and the temperature may be quite normal; so that patients who have remained at home during the first three days will go to their doctor or the hospital with an abundant crop of papules all over the body, but feeling comparatively well, free not only of the fever, but of the headache, lumbar pains, vomiting, and general malaise.

But when the pocks become purulent a fresh access of fever takes place, a *secondary* or *septic fever*, which may be ushered in by chills, or a rigor, and which lasts from three to six or eight days. The temperature rises to 103° or 104°, is mostly remittent in its course, and is accompanied with sleeplessness, headache, and delirium, and a pulse of 100 to 120. All this again subsides as the scabs dry, and the swelling of the skin decreases. With the fall of the scab some patients lose their hair, and even their nails.

Varieties.—Many subdivisions of small-pox have been made: the following are the varieties usually recognised at the present time:—(*a*) ordinary or discrete small-pox, to which the previous description mainly applies; (*b*) confluent small-pox; (*c*) malignant small-pox, the purpura variolosa before mentioned; (*d*) hæmorrhagic small-pox (*variola hæmorrhagica pustulosa*); (*e*) modified small-pox; and (*f*) inoculated small-pox.

(*b*) *Confluent small-pox* is a form in which the eruption is very abundant, and the general illness is correspondingly severe. The initial fever is high, and the temperature does not fall to normal with the appearance of the rash, as it does in mild cases. The rash appears early, even by the second day, and is very abundant; so that on the face, which is most affected, the pustules are closely set, the skin is enormously swollen, and in the stage of suppuration

several pustules coalesce and form irregular and more or less extensive purulent blebs. The implication of the mucous membranes of the nose, throat, and larynx is much more constant and severe. The secondary fever is also high, and is accompanied by prostration, rapid pulse, and delirium or coma. Complications are more frequent and serious, and the mortality is great, death taking place from exhaustion, or hyperpyrexia, or pyæmia.

(*c*) *Malignant small-pox*, or purpura variolosa, in which hæmorrhages appear in the skin within the first forty-eight hours, has been mentioned (*see* p. 72); in another form (*d*) the hæmorrhagic tendency shows itself later. The specific eruption appears, and then hæmorrhage takes place into the papules, or later still into the pustules, or into the skin between the pustules. The petechiæ often occur first on the lower extremities. The mucous membranes are also affected with hæmorrhages, or diphtheritic patches, and bleeding occurs from the nose, lungs, rectum, kidneys, or uterus. The cases are mostly fatal.

(*e*) *Modified small-pox*, often called *varioloid*, occurs for the most part in those who have been vaccinated, but in whom the protection is incomplete, either from deficient vaccination originally, or from the decline of its influence with lapse of time. No broad line of distinction can be drawn between this and the milder forms of discrete variola, but it presents many irregularities. It may be altogether milder, with less fever, and fewer papules; and the eruption may not go beyond the vesicular stage, or even the papular stage. Sometimes the initial fever is severe, with a high temperature, but it is generally of short duration; the secondary fever is very slight, and the eruption is only imperfectly developed. According to some writers, the erythematous initial eruptions are almost confined to these modified cases. Recovery is the rule.

(*f*) When small-pox is *inoculated* a pimple rises on the second day at the seat of the operation; this develops into a vesicle or pustule. The patient then has rigors, swelling in the axillary glands, and some fever; and about the eleventh day the usual eruption of small-pox appears, and passes through its stages. The attack is generally mild, but it is contagious and occasionally fatal.

The **Complications and Sequelæ** are chiefly as follows:—Abscesses and erysipelas, conjunctivitis, and sometimes destruction of the eye from suppurative keratitis; chronic otitis and caries of the bones of the ear; in the respiratory system, bronchitis, broncho-pneumonia, and pleurisy, and the changes in the larynx above described. On the side of the nervous system the following may occur: hemiplegia, probably from arterial thrombosis, myelitis in different forms, occasionally disseminated sclerosis, and multiple neuritis.

Pathology.—Apart from the anatomical characters belonging to the above complications, there is no more to be found in cases dead of small-pox than in other eruptive fevers. In hæmorrhagic cases blood may be found effused into the solid viscera. Examination

of the pustules shows that the process begins with hyperæmia of the papillary layer of the cutis, then the superficial layer of the cuticle is raised from the deeper layers to form a vesicle. The umbilication is sometimes determined by a hair, or the duct of a sweat gland preventing distension at this spot, or merely by cells of the rete Malpighi stretched into a fibre: bands and fibres formed in the same way constitute the septa dividing the vesicle into *loculi*. The pustule becomes hemispherical, in the later stages of suppuration, by the central band or *retinaculum* giving way. Whether the resulting scars are superficial or deep depends upon the extent to which the suppurative process involves the papillary layer of the skin.

Although micrococci have been found in the pus and in various organs, nothing is as yet known of the specific micro-organism of this disease.

Diagnosis.—During epidemics of small-pox the occurrence of shivering, with severe pain in the head and back and vomiting, should make one suspect this disease, but at other times the onset may suggest other acute diseases. The petechial eruptions on the lower parts of the abdomen and in the groins are very characteristic; but the erythematous eruptions may closely simulate scarlatina or measles. The scarlatiniform eruption of variola is most marked on the trunk and lower part of the abdomen or adjacent thigh, and spares the face and neck. It is unaccompanied by inflammation of the throat. The morbilliform eruption is not raised like that of measles. The converse mistake often occurs; that is, measles may be called small-pox. Syphilis also sometimes presents an eruption like it. The variolous eruption is characterised by its hard papules, appearing first on the face, and these are often grouped in twos and threes. The special features of purpura variolosa, the early appearance of the hæmorrhages, and possible death before the papular eruption, should be borne in mind; indeed, a severe hæmorrhagic eruption, coming on rapidly, with the indications of an acute fever, is generally due to small-pox. The diagnosis from varicella is given later (*see* p. 80).

The **Prognosis** has been already indicated. In modified small-pox and the discrete variety it is favourable; in confluent and hæmorrhagic cases unfavourable. Thorough vaccination and re-vaccination diminish to a remarkable extent the severity of the disease; and accordingly prognosis is favourable in proportion to the evidence that these operations have been successfully conducted—*i.e.*, the number and possibly the size of the scars.

Treatment.—The general lines of treatment are the same as for the infectious diseases already described. The patient must be isolated for the sake of others; he must be in bed in a well-ventilated room, and should have abundance of milk and beef-tea, and cooling drinks to quench thirst.

The surface of the body should be sponged with tepid water, and vaseline may be applied where itching is troublesome. The

swelling of the face may be relieved by cold compresses, and the eyelids should be frequently washed, and a little astringent lotion dropped within them. Opium may be given to procure sleep. In the severer cases stimulants may have to be given freely.

Many attempts have been made to prevent the scarring or "pitting," which causes so much disfigurement after a severe attack. Painting the face with iodine, evacuation of the vesicles, and touching with a point of solid silver nitrate, anointing with oil, or carbolised oil, have all been recommended; but it is doubtful if any of the processes is to be relied upon.

If an unvaccinated person has incurred the risk of small-pox he should be vaccinated at once, as it is certain that the disease may be favourably modified by this procedure.

VACCINATION

Prevention of Small-pox.—*Inoculation and Vaccination.*—The observation that small-pox, when conveyed by inoculation of the contents of the vesicle under the skin, produced a milder attack than that commonly conveyed by contagion, led to the use of inoculation as a means of protecting the individual from the more dangerous forms of the disease. Lady Mary Wortley Montagu introduced the custom into England in the early part of the eighteenth century, and her example was widely followed. But a serious disadvantage attached to this proceeding; the small-pox induced by inoculation, though mild, was contagious, and the spread of the disease was thus decidedly favoured. Inoculation consequently fell into disrepute, and finally yielded to vaccination—*i.e.*, the inoculation of *cow-pox*, or *vaccinia*—first practised by Jenner in 1796. He was led to make the experiment from the facts, long observed in dairy farms, that cows were liable to a pustular disease of the udders and teats, which was often accidentally communicated to men and women milking them, and that these persons were subsequently insusceptible to small-pox, either by contagion or by the inoculation then in vogue. Conversely, it was observed that those who had had small-pox did not catch the disease from the cows. Jenner inoculated patients with cow-pox, which produced its characteristic effects, and he subsequently found that certain of these patients were insusceptible to a small-pox virus, which set up typical variola in other unvaccinated persons. The power of vaccination to protect from small-pox has been abundantly proved since then by facts which I need not here repeat.

The cow-pox may be conveyed from man to man by means of the lymph contained in the vesicles a great many times without any very obvious diminution of its power to reproduce the disease and to protect from variola. This arm-to-arm vaccination was in common use until recently, but has now been superseded by the use of lymph obtained direct from the calf previously inoculated with the virus.

Vaccination in Man.—When cow-pox, taken from the disease in man, is inoculated again under the human skin, nothing occurs till the end of the second or the third day, when a papule appears at the seat of inoculation. This increase in size, and on the fourth or fifth day a vesicle forms, which enlarges and forms a circular bleb, flat, or slightly depressed in the centre, and pale gray in colour. On the eighth or ninth day the contents begin to be purulent, and a pink zone of inflammation forms around it. The vesicle becomes more opaque; the redness increases in extent, and is accompanied by induration. The neighbouring lymphatic glands become swollen and tender, and a slight degree of fever and malaise are present at this time. About the tenth or eleventh day the pustule begins to dry, and a brown scab forms during the next few days. The surrounding inflammation subsides, and about the end of the third week the crust falls off, leaving a depressed, pitted, and permanent scar.

The Operation of Vaccination.—The English law requires that all children shall be vaccinated before the age of six months, unless the parent provides a medical certificate that the child is unfit to undergo the operation, or makes a statutory declaration of his conscientious belief that the proceeding will prejudice the child's health. The employment of lymph obtained from vaccine-vesicles produced in the calf has obviated the difficulty of supply from human sources, and has met the objections as to the possible transmission or conveyance of disease in human lymphs.

Its introduction was much facilitated by Copeman's demonstration that by thoroughly incorporating six parts by weight of a 50 per cent. solution in water of chemically pure glycerin with one part of the calf-lymph or vesicle pulp, and afterwards storing the mixture for some weeks prior to use in sealed capillary tubes protected from light, any streptococci or staphylococci existing in the lymph, and even tubercle bacilli if introduced, are completely destroyed. The use of *glycerinated calf-lymph* was recognised in the Vaccination Act of 1898.

The part selected for the operation is generally the outer side of the left arm, near the insertion of the deltoid muscle. The skin is first thoroughly washed and rendered aseptic, and put on the stretch by the use of the left hand. The vaccine may be introduced by puncture or by scratching. If by the former, the sterilised lancet charged with lymph from the capillary tube is inserted obliquely at three, four, or five places from a third to a half inch apart, so as to ensure the introduction of the lymph, if possible, without drawing blood. If by the latter, the skin is scratched in two or three directions at the selected spots, by a sterilised lancet or needle, any blood is wiped away, and the lymph is rubbed in; or a drop of lymph is first placed on each spot, and the skin scratched through it. After the lymph has dried, the part should be protected by a pad of boric lint, or other antiseptic dressing.

Vaccination is, in the vast majority of cases, a perfectly harmless

procedure, but occasional accidents are observed. Erysipelas may attack the wound of vaccination, as it may any other wound by accidental infection; and gangrene has very rarely occurred. There can be also no doubt now that syphilis may be transmitted by human vaccine-lymph, even though it is to the naked eye perfectly clear and free from blood; but this is, of course, obviated by the use of calf-lymph. If human lymph is used, it should be known that the child supplying it is free from any suspicion of disease, and comes of healthy parents. On the other hand, the operation should not be undertaken at all on children who are badly nourished or suffering from eczematous or other eruptions; except in times of epidemics, when the risk from small-pox may overshadow every other consideration of possible aggravation of ill-health already existing. There is no evidence that tubercle can be transmitted by vaccination.

Re-vaccination.—The extent to which the first vaccination is efficient is generally estimated from the number and depth of the scars, and amongst fatal cases in epidemic times an inverse proportion has been shown between the number of the scars and the percentage of fatal cases, the mortality being least in those with four or more scars, greater in those with only one scar, and most of all in those stated to have been vaccinated, but without any visible scar at all. But in any case the protective influence of vaccination has only a limited duration—probably from twelve to fifteen years. It thus becomes desirable that every one should be again vaccinated in childhood or early puberty, and subsequently at any age, if small-pox should become epidemic. According to the extent to which the influence has faded, re-vaccination will have different results. It may fail entirely, or only produce a little local irritation; or it may produce a typical vesicle.

CHICKEN-POX

(*Varicella*)

Chicken-pox is a specific infectious disease characterised by an eruption of vesicles. Although it has often been confounded with small-pox, there can be now no doubt that it is a different disease. Its occurrence is not affected by vaccination, and it cannot be inoculated. Its specific micro-organism has not yet been discovered.

It commonly occurs in children, being conveyed by the air or by clothes; and it very rarely attacks adults.

The period of *incubation* is often fifteen or sixteen days, but may be as short as twelve, or as long as nineteen.

The eruption consists, at first of pink spots or papules, on which, in twelve or twenty-four hours, vesicles form. These are generally tense, hemispherical, and from one-eighth to a quarter of an inch in diameter. At first the fluid is clear and colourless, but it soon becomes opalescent or milky, and then the vesicle shrivels, and a

yellow or brown scab forms, which adheres for a few days, and then separates, leaving a pink stain. The perfectly-formed vesicle is surrounded by an inflammatory zone, which subsides as the vesicle dries. Some of the pocks, but never a large number, result in depressed cicatrices.

The period of invasion is represented by febrile reaction, which is generally very slight; and within twenty-four hours the eruption shows itself—most commonly on the chest, but soon also on the face, trunk, and limbs. The spots are not very numerous, but fresh ones form for two or three days after their first appearance, and altogether they number, as a rule, from 50 to 200. A few vesicles form on the mucous membrane of the mouth, palate, and lips. Whatever fever preceded the vesicles continues for a few hours, or for two or three days; it is generally not above 102°, but may reach 104°. The lymphatic glands of the neck may be enlarged. Death very rarely occurs, but convalescence is not always rapid.

In *varicella gangrenosa* some of the vesicles increase in size, become purulent, form reddish-brown or black scabs under which the skin sloughs, and ultimately leave circular ulcers with clean-cut edges. The child becomes very ill, and death may take place. In *V. bullosa*, large bullæ are found in addition to the usual vesicles.

Diagnosis.—Varicella is sometimes difficult to distinguish from modified small-pox, and in epidemics of the latter it has been found desirable to make varicella "notifiable" (*see* p. 17), so that no case of small-pox may escape the attention of the Sanitary authorities. The chief features of chicken-pox are the early appearance of the rash, the fever simultaneously with the outbreak of the rash rather than before or after, the prevalence of the rash on the trunk, the absence of the shotty feel in the papules, and the clear or milky contents of the vesicles. On the other hand, in small-pox the initial symptoms, headache, backache, and vomiting, are very constant; and the eruption is thick on the face, arms and hands, but much less abundant on the trunk.

Treatment.—Children should be isolated, but confinement to bed is not often necessary. Light diet and attention to the bowels are often all that are required.

MUMPS

(*Specific Parotitis*)

Mumps is a specific contagious disease, of which the essential lesion is an inflammation of the parotid gland.

It occurs mostly in children and young adults; young infants, as well as elderly people, are more rarely affected. Males are more susceptible than females. The infection probably obtains an entry by means of Steno's duct.

Symptoms.—The period of *incubation* varies from fourteen to twenty-five days, and is more often nineteen, twenty, or twenty-one days. The commencement may be shown by slight malaise for a

day or two; but the first symptom is often a feeling of pain and stiffness in the jaw and cheek of one side. Swelling then takes place just beneath the lobule of the ear, so that this is pushed out, and the depression between the jaw and the mastoid process is filled up. The swelling then spreads lower, beneath the ramus of the jaw, and may involve the sublingual and submaxillary glands. After a day or two, the glands of the other side become involved, and thus there is a collar of swelling round the whole jaw from side to side. The swelling is pale, shiny, doughy in consistence, and tender when touched: but suppuration rarely takes place. Internally the tonsils and fauces are somewhat swollen. As a result, the teeth can be separated with great difficulty, and not for more than half an inch or so; and mastication and deglutition are very painful, the pain on movement of the jaw being darting, and lasting for some time. The secretion of saliva may be normal, or increased, or diminished. There is a moderate degree of fever, the temperature rising often to 102°. The illness begins to subside after four to six days, and in another similar period the patient may be quite well. Occasionally the skin over the gland desquamates.

Complications.—As a result of mumps, it happens occasionally that the testicles become inflamed, just as the parotitis is subsiding, *i.e.*, about the seventh or eighth day; but it may be earlier or later than this, and the *orchitis* may occur before the parotitis. It is more common in adults than in boys. The process begins in the epididymis, the testicle swells, and there may be effusion into the tunica vaginalis, and œdema of the scrotum; it is accompanied by a rise in temperature, sometimes considerable. The inflammation subsides in a few days, but it may be followed by a permanent atrophy. More rarely there is double orchitis. In females the mammæ may inflame (*mastitis*) or the external genitals swell, and rarely the ovaries are tender. Mastitis has also been seen in boys. Meningitis, optic neuritis, peripheral neuritis and endocarditis are rare sequelæ. *Pancreatitis* has also been recorded, occurring generally at the end of the first week, and lasting from two to seven days, but it has been known to precede the parotitis. It is shown by pain in the epigastrium and left hypochondrium, tenderness and swelling in the same region, pyrexia, nausea, vomiting, and occasionally by diarrhœa or the passage of fat in the stools.

The **Anatomical Change** in mumps is an inflammatory infiltration, serous and cellular, of the inter-alveolar fibrous tissue of the salivary glands.

The **Diagnosis** presents no difficulties, and the **Prognosis** is favourable.

Treatment.—The patient should be confined to one room, or even to bed, and food must be in a fluid form. Locally, fomentations and opium or belladonna applications generally give relief; and an antiseptic mouth-wash, containing boric acid, should be used frequently. Internally, small doses of potassium citrate or ammonium acetate may be grateful to the patient.

WHOOPING-COUGH

(*Pertussis*)

Whooping-cough is a disease characterised by a peculiar convulsive cough, followed by a long-drawn inspiration through the nearly closed glottis, by which a crowing noise, or "whoop," is produced.

It is contagious, generally requiring rather intimate contact, but sometimes apparently conveyed by clothing, and sometimes certainly by the sputum. Children are very susceptible, and most people have the disease in early life, while it quite rarely attacks adults. A second attack in the same patient is even more rare than in the case of the exanthems. It is most common between the ages of one and eight years, and girls are more liable to it than boys. It occurs in epidemics, but there is not much evidence that such epidemics are determined by climate or weather. It has often been observed that an epidemic of whooping-cough has immediately followed an epidemic of measles.

Symptoms.—The period of *incubation* is about ten days. The first stage is one of bronchial catarrh, which is not always distinguishable from an ordinary catarrh induced by exposure to cold. There are cough, expectoration in children old enough, a few rhonchi in the chest, and slight pyrexia; but sometimes with the cough there is an unusual repetition of the expiratory effort, which may lead to suspicion. This preliminary bronchitis lasts from seven to ten days, and then there is a more or less rapid transition into the whooping stage. First, perhaps, a long-drawn inspiration follows the cough, and then an unmistakable "whoop." But the cough itself is as characteristic as the whoop. The child may be playing with its toys, apparently well, when it suddenly stops, seems distressed for a moment, and then perhaps runs to its mother or nurse. A short cough occurs; this is quickly followed by another and another without any intervening inspiration, each successive cough getting less loud and more stifled until they have mounted up to fifteen or twenty expulsive efforts in the course of seven or ten seconds. Then follows a long-drawn inspiration with loud laryngeal sound, the "whoop"; another burst of short cough succeeds, with another "whoop"; and this sequence may occur once or twice more, with less violence and less noise, until finally a little tough mucus is expectorated, or vomiting takes place. During the coughing efforts, the face becomes congested or cyanosed, the features swollen, the eyes starting from the head, the tongue hanging from the mouth, blood-stained saliva is coughed in all directions, and little relief takes place even from the inspiration, until the final expectoration of mucus or the cessation of the paroxysm. During this time the child is quite given up to the absolutely uncontrollable reflex process; a child in bed, when it feels the attack coming on, will seize the porringer and place it under its mouth, and in another few seconds it will be entirely at

the mercy of the cough, and regardless of what is going on around. As a result of the obstruction to respiration during the coughing efforts, hæmorrhages frequently take place, bleeding from the nose, mouth, or gums, subconjunctival ecchymosis, petechiæ under the skin, and in very rare cases cerebral hæmorrhage. After a time the face often acquires a puffy and bloated appearance from the frequent obstructions to the return of blood to the chest. Sometimes a small ulcer forms on the frænum linguæ, from the pressure of the lower incisor teeth during the cough. The attacks often appear to be spontaneous, but they constantly occur if the child cries or gets in a passion, or even if the child is disturbed, as when the nurse begins taking off the clothes for an examination of the chest. The attacks occur both day and night, and it has generally been noted that they are more frequent in the night hours, between 6 P.M. and 6 A.M., than during the other twelve hours of the day. Observations in the whooping-cough ward of the Evelina Hospital for sick children did not confirm this. The number of paroxysms, which may, as above shown, include three or four actual "whoops," ranges from one to sixty in the twenty-four hours, but it is very rare to have forty attacks, and many cases never reach thirty in the twenty-four hours. In the intervals the child may be perfectly well, and is free from fever, unless there is some complication; the appetite also may be good, and the child soon replaces what he loses by vomiting, which does not, as a rule, occur in more than a small proportion of the paroxysms. This second stage of whooping-cough lasts a variable time, often from three to six weeks, but it may be three months or even more. The attacks gradually get less frequent, until they cease altogether, or as they diminish they may be accompanied with attacks of simple cough, not followed by a whoop, and this may last a few weeks longer. Death rarely occurs directly from the paroxysms; it may do so from prolonged closure of the glottis, or from cerebral hæmorrhage.

Other **Complications and Sequelæ**, however, occur, which make whooping-cough a serious and even dangerous complaint. Amongst the former may be classed *bronchitis*, which may continue throughout, and *broncho-pneumonia*, which is revealed by high fever of remittent type, by crepitant râles or patches of dulness and tubular breathing, and by continued dyspnœa in the intervals between the cough. Often, but not always, the whoop is absent during broncho-pneumonia, as it is if any other febrile complication ensues. General convulsions sometimes occur either as a direct result of the whooping attack; or less commonly as the indication of cerebral hæmorrhage or thrombosis; or, it may be, of the onset of pneumonia. As sequelæ, continued bronchitis, emphysema, and *tuberculosis* of the lungs occasionally occur.

Pathology.—It has long been obvious that whooping-cough must be due to a micro-organism; and in 1906 Bordet discovered a bacillus about the size of the *B. influenzæ*, but longer and plumper; and somewhat resembling it in cultural characters. He finds it best

in the early stages of the disease, in the viscid mucus expectorated apparently from the smaller tubes, and there in almost pure culture. The blood-serum of convalescent cases agglutinates Bordet's bacillus; and vaccines prepared from the bacillus have appeared to influence favourably the course of the disease (Freeman).

While the cough must be due to the secretion set up by the organisms, the whoop is not so readily explained. It is generally thought to be caused by a spasmodic closure of the glottis, but a passive approximation of the cords, or a failure to open freely, when the sudden inspiration takes place would probably account for it.

The **Morbid Anatomy** of pertussis is that of its complications, chiefly broncho-pneumonia. The laryngeal and tracheal mucous membranes are injected, and the bronchial glands are swollen.

Diagnosis.—This mainly depends upon the whoop, on the convulsive character of the cough, and on the regularity of the course from the catarrhal to the convulsive stage. Enlarged bronchial glands may cause a cough something like that of pertussis, but there will be no history of infection, and no whoops; while other symptoms of independent lung diseases may be present. In whooping-cough the leucocytes are increased to 15,000 or 30,000 per cubic mm.; and a differential count gives 60 per cent. of lymphocytes to 40 per cent. of polymorphonuclears, with a few eosinophiles. The lymphocytes are mostly of the small variety, but large lymphocytes are in excess of the normal. These changes occur quite early, and are of use in diagnosis (H. T. Ashby).

The **Prognosis** is to be made from the severity of the complications.

Treatment.—The child should be kept in a warm but well-ventilated room, but confinement to bed is not necessary in an uncomplicated case. A variety of drugs has been used to check the paroxysms of pertussis; the length of the illness may be diminished and the severity reduced by their means. Belladonna is much used in the form of tincture, of which 2 or 3 minims may be given to a child two years old, three times a day, and larger doses to older children. The dose may be cautiously increased up to 10 to 15 minims in a child of five or six. Dilute hydrocyanic acid (1 to 2 minims), chloral (2 to 5 grains), potassium bromide (2 to 5 grains), hydrobromic acid (3 to 10 minims), and antipyrin (2 to 5 grains) are also often used. More recently bromoform (2 to 5 drops mixed with almond oil and mucilage of tragacanth or acacia) and cocaine hydrochlorate ($\frac{1}{15}$ to $\frac{1}{3}$ grain) have been given. Various antiseptic vapours have been used to impregnate the room which the patient occupies, such as carbolic acid, creosote, eucalyptus oil, or sulphurous acid. For the latter purpose, an amount of sulphur equivalent to 10 grains per cubic foot is burnt in the empty and closed room; after five hours the doors and windows are thrown open, and the child sleeps there the same evening. The day nursery is similarly fumigated during the night. Painting the back of the throat or

the glottis with resorcin in 2 or 3 per cent. solution is also said to be of value.

For obstinate cases of cough, after subsidence of the whoop, alum internally is of value (2 to 5 grains), or change of air to the seaside.

The complications must be treated, both as regards drugs and general management, in the same way as they would be apart from pertussis. (*See* Bronchitis and Broncho-pneumonia.)

GLANDULAR FEVER

This complaint, described by Pfeiffer, Park West, Dawson Williams, and others, consists of an inflammatory swelling of the deep cervical and other lymphatic glands associated with fever. It may occur in epidemics, is no doubt infectious, and affects chiefly children under fourteen years of age. It has an incubation period of from five to seven days. The patient is taken suddenly with stiffness in the neck, difficulty of swallowing, and febrile reaction, with anorexia and perhaps vomiting. The fauces are little, if at all, affected, but on the second or third day of pain the cervical glands, and generally those under the sternomastoid muscle and along its anterior border, are found to be enlarged and tender. In another day or two those of the other side are swollen, and the posterior cervical, axillary, and inguinal glands may be also involved. There is generally abdominal pain and tenderness; and the liver, spleen, and mesenteric glands are enlarged. The glands begin to get smaller after from two to five days, and do not suppurate. The temperature may reach 104° on the third day, and it will continue high as long as the glands remain enlarged. Constipation is often troublesome. The disease subsides usually without complications; but nephritis sometimes occurs, and convalescence may be retarded by anæmia. The bacteriology of the disease is at present negative; but a leucocytosis of 18,000 or more, with an increase of the small uninuclear corpuscles, has been described in one epidemic.

The treatment consists in rest, a simple diet, the relief of the constipation by small doses of mercury or salines, and the use of preparations of iron during convalescence.

INFLUENZA

This term, often wrongly applied to any severe nasal catarrh, is the name given to an acute febrile disease which in past times has frequently swept as an epidemic over Europe, but after the violent outbreak of 1847–48 was practically unknown among us until the winter of 1889–90, when the disease again appeared. On this occasion it was first observed in Bokhara in the preceding May; it appeared at St. Petersburg in October, and soon invaded Austria, Germany, France, England, and other European countries, as well as the

United States of America. A few months later it was conveyed to India, Australia, New Zealand, the African Coast, and South America. The disease has again frequently broken out in the British Isles, and of late years has rarely been entirely absent.

Ætiology.—The true epidemic invasions of influenza have always been characterised by the extraordinary rapidity with which the population has been attacked, especially in crowded towns. Hundreds have been struck down at the same time, or within a few days, and this, among other circumstances, led to the view that the disease was not contagious from man to man, but was borne by the air simultaneously to many people. This feature was especially marked in 1889–90. But there is no doubt that the spread of the disease is determined by human intercourse, however sudden the outbreak may seem to have been. Infection takes place by personal contact, and the period of incubation may be as short as a few hours.

Symptoms.—There is the greatest possible variety in the manifestations of influenza. In a large number of cases the symptoms are those of an acute febrile illness, without special determination to any one organ or system of the body. This may be described as the *simple* type, or *simple febrile* type.

The disease begins suddenly with severe frontal headache, pains at the back of the eyes and muscular aching and pains in the muscles of the loins, thighs, calves, and other parts of the body. Rigors are often absent, but the temperature rises within a few hours to 102°, 103°, or 104°. The other accompaniments of fever are present, such as quick pulse, thirst, and scanty, high-coloured urine. The tongue is flabby, tremulous, indented, and covered with a thick white fur. The fauces and tonsils are red; and the breath is offensive. The skin is generally dry, but there are sometimes profuse perspirations. The spleen is sometimes slightly enlarged. The patient is exceedingly ill, restless, sleepless, prostrate and depressed. No other symptoms may appear, and the temperature falls in twenty-four, thirty-six, or forty-eight hours as rapidly as it rose; but the general pains in the limbs continue for some time after the temperature has fallen, and the sense of prostration, which is present from the first, persists for some days after the fever. However, it must be admitted that there is much variety in the course and duration of this group of cases; and that while in some the fever is high, of short duration, and falls rapidly, in others the course is longer, and the fall of temperature more gradual, so that a confusion with other febrile illnesses, such as typhoid fever, is rendered possible. In either case there may be a relapse.

In the *respiratory type* of the disease the commencement presents the same features, namely, fever, headache, pains in the limbs, and prostration; but it is soon seen that the respiratory tract is largely involved. There are rapid breathing, pain in the chest and troublesome cough, and after a time examination of the chest will reveal signs of a bronchitis, such as sibilant or sonorous rhonchi,

and these are accompanied by the expectoration of viscid mucus. Perhaps more often the signs are those of broncho-pneumonia, abundant fine crackling or rustling râles are heard at one or other base, or more extensively over the lungs, confluent or in patches; bronchial breathing is often absent, and resonance may be little impaired. Dyspnœa is pronounced, and the sputum is often blood-stained, abundant, frothy, and not very tenacious. Less frequently, the signs are more like those of a lobar pneumonia; and occasionally pleurisy or empyema may succeed. These cases are severe and often fatal: the course of the temperature is dependent largely upon the progress of the pulmonary lesion. Nasal catarrh, with suffusion of the conjunctivæ, is occasionally a condition of influenza, but both the simple and respiratory forms commonly occur without them.

The *abdominal type* is less frequent, but varies with different epidemics. The patient has abdominal pain, diarrhœa, perhaps some vomiting, and occasionally jaundice. The temperature is often less high than in the preceding forms.

Both the respiratory and the gastro-intestinal symptoms may appear to be rather complications and sequelæ than parts of the original disease; that is, the fever and pains may be present for a few days before either of these systems is manifestly involved. Other systems are also involved more often secondarily or rather late in the history. Sometimes there are attacks of syncope, or collapse, or dyspnœa, or irregular or intermittent pulse, or tachycardia; and the heart may show evidences of dilatation. Hæmorrhages from the different mucous surfaces are sometimes observed.

The nervous system is frequently involved. Drowsiness occurs in early stages; with delirium in severe cases. Later there may be sleeplessness, a persistent neuralgia, or muscular pains. In a large proportion of cases, and without any special localisation of symptoms in the nervous system, there is prolonged weakness of the limbs, inability for physical and mental exertion, and great mental depression lasting for weeks after the beginning of the attack. The skin is occasionally the subject of eruptions in the height of the attack, or a little later. These are mostly in the form of rose-coloured spots, or erythematous rashes like those of measles, scarlatina, or urticaria. In addition there is scarcely any local inflammation that may not in some case or other appear as a sequel of influenza; for instance, otitis, orchitis, peripheral neuritis, phlebitis, parotitis, pericarditis, meningitis, encephalitis, myelitis, conjunctivitis, keratitis, nephritis, arthritis, and lymphadenitis. Amongst functional nervous troubles not hitherto mentioned are loss of taste and smell, and mental breakdown in the form of melancholia or delusional insanity.

Pathology.—As influenza is rarely fatal except through one of its inflammatory complications, its morbid anatomy is generally comprised in that of the lesion, such as pneumonia, which has immediately caused death. The minute bacillus, discovered by Pfeiffer in the sputum of influenza patients, but rarely in the blood,

is no doubt the specific microbe of the disease. Streptococci, staphylococci, and pneumococci, are also found in connection with the secondary lesions. The *B. influenzæ* is negative to Gram's stain, and grows best on media containing hæmoglobin or blood. It is probable that there are two or more strains, possessing different pathogenic properties.

Diagnosis.—The great variety that influenza presents will lead to its being diagnosed in the early days of an illness, when further acquaintance with the case may show it to be some other febrile complaint, such as pneumonia, and especially enteric fever (*see* p. 51). The very sudden onset, the local pains, and the short fever are the chief distinguishing points of influenza; in some cases with pulmonary or bronchial lesions the presence of Pfeiffer's bacillus may be demonstrated in the sputum.

Treatment.—The patient should save his strength by at once taking to his bed. In the early stages the severe pains call for treatment, and may be met by sodium salicylate (10 to 15 grains every four or six hours), or by aspirin (7 to 10 grains), or by phenacetin (5 or 6 grains). The great tendency to prostration after the illness makes it necessary to give these drugs with caution. Instead of them, salines (10 or 15 grains of potassium citrate, or half an ounce of liquor ammonii acetatis) may be given in the early stages, combined with expectorants, if there be much bronchial complication (ammonium carbonate, 3 to 5 grains, or tincture of senega, ½ to 1 drachm). As the fever subsides, most cases require a tonic regimen. Quinine and nux vomica are especially useful; and in older patients stimulants are also needed.

The local manifestations of the disease require to be treated as they would be if arising under other circumstances.

CEREBRO-SPINAL FEVER

(*Epidemic Cerebro-Spinal Meningitis*)

This disease was first recognised at Geneva in 1805. Since 1860 it has been prevalent in the United States and in Germany. In 1846 it appeared in Ireland, and again in a severe form in 1866–68; and in 1906–8 some hundreds of cases occurred in Glasgow and in other towns in Scotland, and a few in London.

Ætiology.—Very little is known of the conditions determining the transmission of the infection. The entrance of the virus by the nose, ear, and throat has been suggested, but direct contagion from man to man, or by means of clothes, has often failed to occur, when it might have been expected. The young are most often the subjects of the disease, 80 per cent. of cases being under sixteen years of age, and only about 5 per cent. over twenty-five. The sexes are attacked nearly equally.

Symptoms and Course.—There are in a few cases slight premonitory symptoms, such as headache, nausea, or malaise; but

mostly the disease begins suddenly with severe headache, and sometimes a rigor, so that the patient has to give up at once, and suffers also from pain in the back and limbs, vomiting, and fever. The headache is chiefly occipital, but may be frontal or temporal; with it is stiffness of the muscles of the back of the neck, which may be shown by attempting to bend the head forwards, and often the head is drawn back by the contraction of the deep muscles: the dorsal and lumbar muscles may be similarly affected, so that the back is kept straight (*orthotonus*), or even arched with the concavity backwards (*opisthotonus*); and sometimes the legs and arms are flexed in tonic spasm. Pains frequently extend down into the muscles of the lower extremities, and cutaneous hyperæsthesia may be also present. The knee-jerks are often active, but may be absent; Kernig's sign is frequently observed, and less often Babinski's sign (*see* pp. 207, 204). In addition to these symptoms, referable to irritation of the roots of the spinal nerves, there are others due to the implication of the cranial nerves. These may be, in different cases, ptosis, or strabismus; contraction or dilatation, or inequality of the pupils; or contraction of the facial muscles; but trismus is rare. Optic neuritis and purulent irido-choroiditis occur, and conjunctivitis and keratitis, probably from external irritation. Pain in the ear, tinnitus and defective hearing are not uncommon, and suppuration of the labyrinth or of the tympanum may occur. Deficiency of the sense of smell has been noted. Drowsiness, delirium, and coma, sometimes with Cheyne-Stokes breathing, or convulsions, supervene in due course; and death takes place with varying rapidity in different cases. Fever is present from the first, but it runs no regular course; it is remittent or intermittent, perhaps normal for a day or two, and then rising to 102° or 103°, seldom exceeding 104°. Occasionally the fatal termination is preceded by a temperature of 108° or 109°. With recovery the temperature falls slowly and irregularly. An important feature of epidemic meningitis is the occurrence of cutaneous eruptions, of which herpes facialis is the most frequent. It appears early in the illness, as frequently in severe as in mild cases, and in more than half altogether. It often covers a large part of the face, and may be bilateral; and sometimes it affects the trunk or limbs. Urticaria, erythema, and purpura also often occur. All these eruptions may appear at the same time, and they are often symmetrically arranged. Sometimes the joints are inflamed, hot, red, painful, and swollen—a condition which generally subsides, but may go on to suppuration. The abdomen is often retracted; the spleen is not often enlarged. There is not infrequently polyuria, and the urine may contain a little albumin, or a trace of sugar. A leucocytosis of from 15,000 to 60,000 is constant, and is nearly always polymorphonuclear: only occasionally in infants and young children there is a lymphocytosis. The pulse is generally quick, and perhaps irregular. The fluid drawn off by a lumbar puncture (*see* p. 213) is usually turbid and may be purulent; it deposits polymorphonuclear leucocytes, and cultures yield in most

instances the *diplococcus meningitidis intracellularis* of Weichselbaum, sometimes the pneumococcus, or staphylococcus aureus.

The mortality varies from 30 to 70 per cent. in different epidemics

Varieties.—Some cases are fatal in a few hours or days (*foudroyant*, fulminant or explosive); others, on the contrary, are abortive, getting well rapidly in a few days; the majority last from two to four weeks. Remittent and intermittent forms are recognised, in which the fever is much less or absent for periods of two or three days at a time; and a "typhoid" form with muttering delirium, dry brown tongue, involuntary evacuations, and bed-sores.

It is now generally agreed that we must regard as a variety of epidemic cerebro-spinal meningitis the *posterior basic meningitis* of infants, which, first described by Gee and Barlow under the name of "cervical opisthotonos," occurs sporadically in infants and young children.* The infective agent, which is found in the meninges, and in the cerebro-spinal fluid, appears to be identical with the *meningococcus* of the epidemic form. It is probable that infection takes place through the naso-pharynx, the Eustachian tube, and the tympanic cavity.

Retraction of the head may be the first change noticed, or there may be vomiting, convulsions, drowsiness, or screaming. The retraction of the head is accompanied by retraction of the spine and by rigidity of the limbs, either in flexion or extension. There is often strabismus, and occasionally nystagmus and retraction of the upper eyelid; optic neuritis is uncommon, but the children are often blind. Clonic convulsions are not very frequent, nor is actual paralysis of the limbs. The temperature is often febrile, and in some cases sudden rises to 103° or 104°, followed by a fall in a few hours, occur daily or every other day, or more irregularly.

The illness often lasts several weeks, during which the children emaciate; and in many cases death occurs. A few cases recover completely, but some suffer the same sequelæ as occur in the epidemic varieties.

Sequelæ.—Deafness is most common from the lesion of the internal and middle ear, and if both ears are destroyed in very young children deaf-mutism necessarily results. Vision is also often impaired. Chronic hydrocephalus is another result of the preceding inflammation, and may have for its symptoms headache, convulsions, mental deficiency, and weakness of the limbs. Hemiplegia, paraplegia, and aphasia also occur, but are generally of a transitory nature, if first appearing within a short time of the illness.

Morbid Anatomy.—There is an acute leptomeningitis of the brain and spinal cord. The pus and lymph are most abundant on the convexity of the brain, along the larger blood-vessels and in the fissures. In the spinal cord the posterior surface is more

* The London County Council accepts this view, and requires the notification of cases of posterior basal meningitis.

affected than the anterior, and the lumbar region more than the other parts. The ventricles of the brain contain turbid serum or pus. Punctiform hæmorrhages, accumulations of leucocytes, or actual abscesses are found in the cortex of the brain. Other changes found are congestion of the lungs, liver, spleen, and kidneys, fatty degeneration of the renal epithelium, and granular degeneration of the voluntary muscular fibres; sometimes ecchymosis of the pericardium and pleura, and suppuration of the joints.

In the sporadic forms of posterior basic meningitis the meningeal exudation is one of lymph, rather than pus, and covers the base of the brain (pons, medulla, and cerebellum) posterior to the optic chiasma, and sometimes extends to the cervical spinal cord. In cases of long duration pronounced hydrocephalus is present from obstruction of the foramina (of Majendie and others).

The specific organism is a diplococcus (*D. meningitidis intracellularis* of Weichselbaum), which is seen in the polymorphonuclear leucocytes of the meningeal exudations, but also free: it is also found sometimes in the blood, in pus from the joints, in pneumonic foci in the lungs, and in the nasal mucus.

Diagnosis.—This is not difficult in the course of an epidemic. The characteristic features are the sudden onset, the headache, vomiting, pain in the back and limbs, stiff neck, and the herpes labialis. The purpuric eruption also seems to distinguish it from other forms of meningitis, tubercular and suppurative, which must always be carefully considered. The fact that it occasionally complicates pneumonia must be remembered, but must not have too much weight in quite young children, in whom retracted head and convulsions often occur from pneumonia alone. Obscure toxic conditions, such as those arising from some kinds of food-poisoning, have been mistaken for the disease. If the general conditions, with the epidemic associations, and perhaps Kernig's sign, do not suffice for a diagnosis, lumbar puncture will generally yield a fluid from which cultures of the specific organism may be made. The same procedure will be of assistance in distinguishing the posterior basal variety from tubercular meningitis, which may resemble it closely.

Treatment.—A serum treatment has been extensively tried in the epidemic disease, and with a certain measure of success. Several sera have been employed, amongst which Flexner's appears to have reduced the mortality to 25 per cent. The serum is injected preferably into the spinal canal after lumbar puncture, in doses of 30 c.c. every day or every two or three days, according to the severity of the case. Lumbar puncture alone may relieve by reduction of pressure. Treatment other than this is conducted on the same principles as that of other acute diseases. High temperatures may be reduced by ice, or cold sponging; and severe pains may sometimes need the careful use of morphia.

Sporadic cases of posterior basal meningitis may be treated with mercury and potassium iodide. Removal of the intracranial fluid

through the subcerebellar space (basal drainage) and by lumbar puncture has been of use in some cases.

MALARIAL FEVERS

Especially in marshy districts, but also in other localities possessing special features, certain diseases occur endemically, which are known from their clinical characters as *intermittent* or *remittent* fevers, and from their source as *malarial, paludal,* and *marsh* fevers. The word *malaria* (*mala aria,* bad air) was also used to indicate the virus or infective agent before it was known that this was an organism invading the blood-corpuscles and conveyed from man to man by mosquitoes. It is obviously now a misnomer.

The milder forms of the disease which occur in most temperate countries are the *intermittent* fevers (known in England as *ague*), characterised by the periodical recurrence of febrile attacks, separated by intervals of comparative health. The attacks last a few hours, and recur daily (*quotidian* fevers), or every other day (*tertian* fevers, the second attack occurring on the *third* day of the illness), or every third day (*quartan* fevers), and the subsidence of the temperature to the normal after each attack constitutes the intermission from which their name is taken. Tertian ague is more common in temperate climates; quartan is the least frequent form, but it is common in certain parts of Italy and of India. In the hotter parts of the temperate zone, and in tropical countries, the attacks are often more irregular in their occurrence, and of longer duration, with shorter intervals between them; or the temperature fails to reach the normal between the attacks, so that the fever is only *remittent* instead of being intermittent (*see* p. 25), or the temperature is constantly so high that the fever is *continuous.* These forms are more severe than the simple intermittent, and form the *æstivo-autumnal* fevers of the Italians, and the *remittent, continuous* and *malignant* varieties of the tropics.

Ætiology.—These fevers are due to the action of one or more micro-organisms which are contained within the red corpuscles; and the organisms are conveyed from man to man by certain varieties of mosquito, in the bodies of which they pass through one stage of their development. The ætiology of malarial fevers is therefore largely explained by the life-history of the mosquito. Where the mosquito can breed and flourish, and where surface water is provided for the growth of their larvæ, malaria may occur; where mosquitoes cannot breed, malaria will be absent. Even in mosquito-breeding countries or districts those who can protect themselves from the bite of the insect will escape the disease. Though not peculiar to tropical countries—indeed, it was once prevalent in England—the disease is more frequent where the atmospheric temperature is high; and it is practically confined within the latitudes of 63° N. and 57° S.

The other essential condition, indicated in the names *paludal*

fever and *marsh fever*, is the presence of extensive areas of partially submerged land, marshes, ponds, or rivers, with more or less abundant vegetation. The disease is endemic in the flat districts of Holland and North Germany, on the west coast of Italy, and in parts of Greece, on the west coast of Africa, in many parts of India, especially along the Ganges and the Indus, and also in parts of China and Persia.

The parts of England formerly subject to it were the borders of the Thames in Kent and Essex, and the fen districts of Cambridgeshire and Lincolnshire. Though these are all low-lying districts, and salt-water marshes are quite as favourable to its occurrence as fresh-water areas, malaria also occurs at high levels in some parts of the world, as, for instance, in the Apennines at a height of 1100 feet above sea-level, in the Pyrenees at 5000 feet, and in Peru at 11,000 feet.

No race is quite immune, though negroes appear to be less susceptible than white men. The disease may be contracted at all ages. People in ill health are more liable to it, as well as those exposed to damp, to cooling influences, or to excessive heat of the sun, and those who indulge in immoderate eating or drinking.

Malarial infection is not always ended by the cure of the first outbreak, and those who have suffered in malarial districts may, long after removal, be again attacked by fever on the occurrence of slight causes. Moreover, in badly infected districts, such as some in India, Italy and Greece, it is found that a large proportion of the population are anæmic and out of health; and as many as 30 per cent. of the children have in some instances been found to be infected, as shown either by the discovery of the malarial organisms in the blood, or by the existence of an enlarged spleen. Manson, however, believes that infection does not persist after three years from the date of invasion. The organism may be latent in the system, as it has often been observed that a person may be a short time in an ague district, leave it without having an attack, and afterwards, in a perfectly healthy locality, develop the disease.

The Malarial Parasites.—The micro-organisms of malaria were first described by Laveran, and are now known as members of the family *Hæmamœbidæ*. Of these at least three occur in man, and others in similar diseases in birds. These three are the parasite of tertian fever (*Hæmamœba vivax*), the parasite of quartan fever (*Hæmamœba malariæ*), and the parasite of the malignant fevers (*Hæmomenas præcox*). The last can be divided into two varieties, tertian and quotidian.

The first and second of these organisms are protoplasmic bodies, which exhibit amœboid movements, and are first seen in the early part of the intermission within the red corpuscles, looking like clear spaces. In the course of a few hours they enlarge and occupy more and more of the corpuscle; and granules of pigment accumulate in their interior. Then the pigment aggregates

towards the centre of the organism, or *amœbula*,* and a process of division, or *segmentation*, takes place, so that the amœbula breaks up into from six to fifteen or twenty smaller clear bodies, *spores* or *enhæmospores*; and, at the same time, both these and the pigment granules become free in the blood plasma. This process of segmentation corresponds with the onset of the paroxysm of ague; and during its course, while some of the spores are no doubt included in and destroyed by leucocytes, others enter into fresh red corpuscles, and start a new cycle of events by enlarging to form amœbulæ, which again break up into enhæmospores.

The quartan parasite is slower in its development, taking three days to fill the corpuscle and undergo segmentation, whereas the tertian parasite takes only two days; and this difference accounts for the difference between the lengths of the intervals in the quartan and tertian fevers. Moreover, the quartan parasite has a more distinct outline, never so completely fills the corpuscle (which is otherwise but little changed), presents coarse granules of dark brown or black pigment, has slower amœboid movements, and breaks up into from six to twelve segments. On the other hand, the tertian parasite has a less defined outline, causes swelling and pallor of the corpuscle, has fine yellowish-brown pigment-granules, more active amœboid movements, and divides into from fifteen to twenty spores.

There may, however, be two crops or sets of tertian parasites, which segment or sporulate on alternate days, when the fever will be recognised as one of *quotidian* or *double tertian* type: and there may be two sets of quartan parasites maturing on different days, producing a *double quartan* fever; or three sets, producing a *triple quartan*, or, since every day is involved, another *quotidian* type.

The malignant or æstivo-autumnal parasite begins its intra-corpuscular life like the preceding, but is often seen as a small ring-like body one-seventh of the diameter of the red corpuscle, with high refractive power, and containing less pigment than the other forms. Its period of development is irregular, varying from twenty-four to forty-eight hours, and in its later stages (segmentation) it is not found in the peripheral blood-vessels, but exists only in the internal organs (*e.g.*, spleen and bone-marrow). After these fevers have lasted some days or a week, *crescentic* bodies are found, each being a colourless, transparent, immobile mass, with a few pigment granules in the interior, longer than the normal diameter of the red corpuscle which appears to be stretched over it.

Relation of the Mosquito to Malarial Parasites.—The mosquitoes concerned in malarial infection are the *Anopheles claviger*, vel *maculipennis*, and some other species of the genus *Anopheles*. They not only transfer the malarial organisms from one human individual to another, but they allow in the interior of their bodies a true sexual process to take place, quite different from anything that occurs in the human blood.

If the blood from a pernicious form of fever containing crescentic

* According to the nomenclature proposed by Professor Sir Ray Lankester.

bodies (*gametocytes*, or *crescent spheres* *) be examined, these bodies will be found after an interval of some minutes to alter their shape to that of a sphere, and to throw out suddenly three or four fine filaments, or *flagella* (*microgametocytes*, or *spermatozoa*), which perform lashing movements, and ultimately separate from the sphere and move freely in the blood. Others of the crescents are converted into granular spheres without flagella (*macrogametes*). The liberated spermatozoa approach the granular spheres, enter their substance, and thus impregnate them. As a result, the spheres become elongated bodies with a pointed extremity, and are in a position to penetrate other substances: they are called *zygotes*, or *vermicules*, or *ookinetes*. These changes take place in the mosquito's stomach. The zygotes then penetrate the wall of the stomach, where they have been observed, and where they enlarge to a diameter of 60μ or 80μ, so as to project into the body cavity of the animal. In the interior of the zygotes, which, having reached the outside of the stomach, now become spherical *oocysts*, are formed a vast number of minute rods (*sporozoites*, or *exotospores* *) which are discharged by the bursting of the zygote or oocyst into the blood of the mosquito. Thence they reach the cells of the salivary gland at the base of the proboscis, from which they are carried during the process of puncture into the blood of the next human being attacked by the insect. These exotospores enter the human corpuscles, and so give rise to the amœboid bodies, or amœbulæ, first mentioned.

Symptoms of Intermittent Fever.—The period of *incubation* is from three to twelve days, shorter in the irregular, longer in the regular forms; but it may break out almost immediately on exposure. In some cases there are *prodromata*, consisting of malaise, headache, pains in the limbs, epigastric fulness, nausea, and slight chills or flushes.

An attack of malaria or ague consists of three stages: the *cold* stage, the *hot* stage, and the *sweating* stage.

The Cold Stage.—The patient feels tired and listless: has headache and pain in the back and loins, then feels chilly, and the *rigor* begins. He generally lies curled up in bed, shivering all over, and his teeth chattering. The face is blue and pinched, the nose and ends of the fingers livid, and the skin in a condition of *cutis anserina*. The pulse is small, hard, frequent, and irregular; the breathing quick and shallow. The surface is actually cold, but the thermometer, placed in the mouth, rectum, or axilla, will show that the temperature is already considerably above normal. Indeed, it commences to rise some minutes (or even one or two hours) before the rigor; but the sensation of cold and the actual cold of the surface are due to contraction of the superficial vessels. The urine is abundant, clear, and of low density. This stage lasts from half an hour to two hours, and the axillary temperature rises rapidly, attaining to a height of 105°, 106°, or even more, towards the end of the period.

* Sir Ray Lankester.

The Hot Stage begins with a sense of warmth diffusing itself over the body, and the surface, hitherto cold, becomes intensely hot. The temperature in the axilla rises still a little higher than it was at the end of the cold stage; the arteries are relaxed, the pulse becomes quick, full, and hard; the carotids throb, the face is flushed, and the head aches. There is a tendency in some cases to stupor or delirium. The urine during this stage is scanty, dark, and of high density. There is often an eruption of herpes about the mouth. This stage lasts from three to four hours.

The Sweating Stage.—The skin, hitherto dry, now gradually becomes moist. Sweating begins first on the face, and then spreads to the rest of the body, and continues profusely from one to two or three hours, the pains and discomfort of the hot stage are relieved, the pulse becomes softer and slower, and the tongue moist. The temperature falls at first slowly, then more rapidly, until the normal is reached; and, finally, with the subsidence of the temperature the sweating ceases, and there is a return to the preceding state of health. During this stage the urine is of high density, and deposits a sediment of urates.

When the attack is over the patient feels perfectly well; but after an interval determined by the nature and number of the parasites, he is seized with another similar paroxysm.

The attacks are not always complete. There may be only slight chill, but a hot stage of two or three hours, with no sweating; or there may be chill, with sweating, but no hot stage; or cold and hot stages may be absent, and sweating, with a slight rise of temperature, may alone represent the attack.

In ordinary cases the spleen enlarges during the cold and hot stages, so that its area of dulness is increased, and it may be felt below the costal margin. The blood, in addition to the presence of amœbæ, shows a diminution of hæmoglobin and of both red and white corpuscles. Of the latter, the polymorphonuclear leucocytes and lymphocytes suffer most, while there is a relative increase of the large uninuclear cells. This condition persists for some weeks after the attack.

The attacks take place mostly in the morning or noon hours, the tertian especially at noon; succeeding attacks are, however, not always at the same hour, but in some cases may get earlier and earlier—in others later and later. The former are said to *anticipate*, the latter to *postpone*.

Ordinary attacks of tertian fever are not generally fatal, though death may happen in very young or in old people, or in those debilitated by previous illnesses. If the spleen is very much distended it may rupture spontaneously or after injury, and the blood will escape into the peritoneal cavity; or hæmorrhage may occur in its substance, followed by suppuration and rupture of the abscess into the peritoneum.

The complications of simple tertian fever are not numerous.

Bronchial catarrh, epistaxis, and albuminuria occur occasionally; neuralgia, especially of the supraorbital nerve, is not infrequent.

Malignant Tertian Fever, Æstivo-Autumnal, Irregular, Remittent and Continuous Fevers.—These severer forms of fever are due mainly to the malignant tertian parasites, and present features of irregularity and continuance which are probably explained by the variable periods of maturation of the parasites, and by multiple infections. The fever may be intermittent, of quartan, tertian, or quotidian type, but the febrile attack is often very long, and the interval short. The attacks have a tendency to *anticipate*, and thus a tertian becomes a quotidian; and ultimately the interval may be lost or the fever becomes remittent. Or the cases are remittent from the first, or the fever is continuous. Rigors are much less common than in the benign forms; and jaundice, nausea, vomiting, and diarrhœa are not infrequent. Thus the slighter forms are generally known as bilious and gastric remittents; the more severe forms, with continuous high temperature, bear a close resemblance to enteric fever, and are often spoken of as *typho-malarial*. These forms of fever may last from a few days to two or three weeks; but are not infrequently fatal, with such symptoms as coma, delirium, fever, severe gastro-intestinal disturbance, albuminuria, hæmorrhages from various parts, and collapse. During recovery the remissions become more marked, until actual intermissions may occur.

Sometimes these dangerous symptoms will suddenly occur in the course of what appears to be an ordinary tertian illness, and the case develops into a *pernicious* form, characterised by the profound implication of one organ or system, such as the bowels, nervous system, or lungs. These are said to occur only as a result of the æstivo-autumnal parasite, and no doubt arise from vast numbers of the parasites invading the vessels of the organs concerned.

Among forms involving the nervous system may be mentioned a *comatose* form: there are marked headache, dizziness, apathy, or even drowsiness in the cold stage: and these pass into complete unconsciousness in the hot stage, with rapid, stertorous breathing, wide, immovable pupils, and limbs completely relaxed. The patient may lie thus for ten or twelve hours, or more, and then gradually recover consciousness in the sweating stage. In other cases there may be *delirium*, or violent maniacal attacks, with screaming, and hallucinations. The patient may sink into coma and die, or gradually fall into sleep, from which he recovers. Cases are described in which the patient's condition simulates death, with arrested respiration, and imperceptible pulse or heart-beat Different forms of *convulsions* also occur sometimes, and sometimes *hyperpyrexia* is seen, so that the case may be mistaken for heat-stroke. On the other hand, an *algide* form is also described, in which extreme collapse occurs during or after the hot stage, the body becoming intensely cold—the temperature in the mouth 86° to 88°, and in the axilla only 84°. The surface is pale or livid, and covered with

sweat. There are vomiting and diarrhœa, shallow or slow respiration, and feeble, hoarse voice. These cases are generally fatal. Other cases are complicated with *dysenteric* attacks, or hæmorrhage from the stomach or bowels; or, on the other hand, *pneumonia, pleurisy,* or *jaundice.* In these last cases an icteric tinge of the conjunctiva may show itself before the attack; if not, it appears during the cold stage, and the yellow colour spreads over the whole body. There is intense nausea, with bilious vomiting, and the urine is scanty and bile-stained. The symptoms are all aggravated in the hot stage, the profuse sweat of the third stage is bile-stained like the urine, the jaundice persists during the interval, and there is a great tendency for the fever to lose its intermissions and become remittent. Death may take place in the hot stage.

Blackwater Fever; Bilious Hæmoglobinuric Fever.—In certain parts of the world (the west coast of Africa, Madagascar, and the opposite east coast of Africa, Siam, New Guinea, the Southern United States, Central America, and Venezuela, and Guiana), which are mostly tropical, and all within malarial areas, occurs the disorder known by the above names: and for the most part it occurs to those who have been some time in the locality, and who have had true malarial attacks previously. After a few prodromal symptoms, malaise, pains in the limbs and head, there is a definite rigor, followed by vomiting of bile, and soon a quantity of pink, red, or black urine is passed, the colour of which is due to hæmoglobin, and not to sound blood-corpuscles. It is acid, albuminous (to the extent of $\frac{1}{2}$ to 2 per mille by Esbach's test), and deposits a dirty brown sediment of epithelium, granular débris, and hyaline casts. With the rigor the temperature rises to 104° or more, but falls somewhat after a few hours, rising again with rigor, and this may be repeated again. The patient is more or less deeply jaundiced, and the liver and spleen enlarge and are tender. In favourable cases the symptoms last about a week; in fatal cases, vomiting continues, the urine becomes more albuminous and scanty; suppression may occur, and death follows with coma or collapse. The mortality is from 16 to 50 per cent. Though it is almost certainly a malarial affection, the parasites have often been sought in vain; but this may be accounted for by the destruction of the red blood-corpuscles, of which the hæmoglobinuria is evidence. Indeed they are frequently found if the blood is examined the day before the onset of the symptoms and on the day of the actual attack; the leucocytes, moreover, are pigmented and the large uninuclears are found in the numbers characteristic of malaria (*see* p. 100). Many observers regard quinine as an exciting cause, but the symptoms may arise otherwise sometimes; and it is certainly not a cause apart from malaria.

Malarial Cachexia.—In those who have had repeated attacks of malaria, and in some who have resided in malarial districts without developing such attacks, as well as in the natives, children and adults, of badly affected areas, the bodily health is seriously affected.

The most prominent feature is *anæmia*, causing a sallow, earthy look, with pallor of the lips; it is due to the destruction of the blood-corpuscle by the parasite, and the conversion of the hæmoglobin into black pigment. There are also numerous functional disturbances, dizziness, loss of appetite, digestive disorders, pains in the joints and muscles, lassitude, and indisposition for exertion. In severer forms there is ascites, œdema, or hæmorrhage, but no fever. The spleen in these cases is enlarged, reaching even below the level of the umbilicus, and forward to the middle line, constituting the *ague-cake* of early English writers. It is hard, and often tender. The liver may also be enlarged.

Morbid Anatomy.—In fatal cases there are punctiform hæmorrhages of the meninges and white substance of the brain; the capillaries contain infected red cells, and their endothelium may be fattily degenerated. In the enlarged spleen the trabeculæ of the pulp are distended with infected red cells, but the Malpighian corpuscles are not pigmented. In the liver the endothelium of the capillaries is swollen and pigmented. The liver cells contain hæmosiderin, most abundantly round the central vein. In the bone-marrow there are parasites and melanin, both free and within the large uninuclear leucocytes, and in macrophages. Crescents are often found here, when scanty elsewhere. In long-standing cases the yellow marrow may become red.

In malaria with intestinal symptoms parasites are abundant in the capillaries of the villi.

In old cases, and in malarial cachexia, the spleen has become more fibrous, firm, tough, and pigmented, especially in the connective tissue surrounding the follicles; often with a thickened capsule, adherent to surrounding parts, presenting infarcts, and in long-standing cachexia perhaps lardaceous.

The blood may contain brown or black pigment granules, either free or within the white blood-cells; and this pigment, found also in the spleen, liver, brain, kidneys, heart, and in the lymphatic glands, and marrow of the bones, gives a slaty or dark gray colour to the various tissues. The condition is described as *melanæmia*. The liver is pigmented, mainly in the periphery of the lobules; the capillaries are dilated, and their epithelium is pigmented. The hepatic cells are atrophied. The marrow of the long bones is usually red; and normoblasts are common.

The **Diagnosis** of intermittent fevers is generally easy; the attacks are often distinctive in themselves, and their nature is confirmed by the recurrence at regular intervals, provided that the intervals represent a tertian or a quartan form. Daily rigors are less to be trusted, as they occur in various septic conditions, such as *pyæmia, abscess of the liver, and malignant endocarditis*. The pyrexial attacks in other forms of suppuration and in phthisis may simulate ague; on the other hand, the rigors in these complaints are quite irregular. If treated with quinine, on the supposition that they are malarial, no result will be obtained, whereas malaria yields to an

adequate dosage with this drug. In the severe forms of malarial fever, the nature may be overlooked from the prominence of some local disorder, and from the slight development of the febrile characters; thus the comatose form may be mistaken for apoplexy, and others for pneumonia or cholera. The resemblance of the continuous forms to typhoid has already been mentioned. On the other hand there are *latent infections*, which do not develop sufficiently to produce the typical outbreak. In all these cases the diagnosis can be made by an examination of the blood. A drop of blood obtained from the finger, or in special cases from the spleen, and placed under a cover glass, may be examined with a $\frac{1}{12}$-inch oil immersion lens; or films (*see* Examination of the Blood) may be dried and fixed by alcohol and ether (not by heat), stained and mounted in balsam. The best stains are methylene blue; or a mixture of 30 c.c. concentrated alcoholic solution of methylene blue with 100 c.c. liq. potassæ (1 in 10,000); or a mixture of 2 per cent. aqueous solution methylene blue with an equal quantity of 5 per cent. solution of borax; or diluted carbol-thionin blue; or Romanowsky's stain (prepared from methylene blue and eosin). Assistance can be also derived from enumeration of the leucocytes: generally they are in excess of the normal during the rigor and fever, and then fall below the normal (leucopenia) until the next attack. A differential count shows a great increase of the large uninuclears, especially during the apyrexial period, and a rise of these corpuscles to 20 per cent. of the total leucocytes at this period is strong evidence of malaria in otherwise doubtful cases (Stephens). The leucocytes, especially the uninuclears, are pigmented.

Treatment.—Quinine is the remedy which, in the large majority of cases, effectually controls malaria, and is also used to prevent its occurrence in those exposed to the risk of infection. For the latter purpose the sulphate of quinine may be given in doses of 2 to 5 grains three times daily.

If there are indications of the onset of the disease, 10 grains may be given every night for three or four nights, and will sometimes prevent the outbreak. In the developed disease, 20 to 30 grains should be given in the day, and it is generally thought that this amount is better given in two or three large doses than in smaller quantities at frequent intervals. The last big dose should be given about six hours before the next expected attack. In the severe or pernicious cases still larger quantities of quinine may be necessary, and no prominence of the local symptom should lead one to abstain from its use. If quinine is vomited it may be given with a little opium, or may be injected per rectum, or subcutaneously, or, better still, into the substance of the gluteal muscles. For the last two methods, the acid quinine hydrobromide, which is soluble in 6 parts of water, and is unirritating, is especially suitable. The dose is 2 or 3 grains for ordinary cases, and 5, 10, or 15 grains for pernicious forms.

In a certain number of cases quinine does not completely cure, and arsenic in doses of from 8 to 12 minims of the liquor two or three times daily has been found useful; it is more useful in the chronic form. Methylene blue in doses of 3 to 5 grains in gelatine capsules, three or four times a day, is also recommended; and eucalyptus extract, the sulphate of beberine, and piperine have also been used.

During the attack little can be done but minister to the comfort of the patient and relieve symptoms. In the cold stage the patient must be kept warm by sufficient clothing, and by hot bottles to the feet; and he may be ordered some diffusible stimulant if there is a tendency to collapse. In the hot stage the coverings will have to be removed, the body may be sponged with cold or tepid water, and thirst satisfied with cooling drinks.

In blackwater fever quinine is not desirable unless malarial parasites are present, when it should be given for their destruction (Manson, Crosse); and preferably as hydrobromate or di-hydrochloride in 2 or 3 grain doses frequently. Intramuscular injections may also be employed. Otherwise the treatment is chiefly symptomatic; Cantlie recommends turpentine, and Hearsey advises small doses of corrosive sublimate, and sodium bicarbonate.

Malarial cachexia requires removal from the infected district, and internally the use of iron and arsenic; Maclean recommends the baths of Carlsbad and Homburg. Enlarged spleen is said to be amenable to the cold douche directed upon it, or to the ointment of red iodide of mercury smeared over the surface, which is then exposed to the fire till it begins to smart. The continuous current of galvanism may also be employed. Internally quinine, iron, and iodide of potassium should be given, or the phosphates of iron, quinine, and strychnia.

Prevention.—This can be furthered by (1) the drainage of malarious areas; ague has thus almost entirely disappeared from the British Isles; (2) the extermination of the mosquito; this has been attempted by pouring kerosene upon pools in which mosquitoes breed, so as to destroy the eggs and larvæ: one ounce is sufficient for fifteen square feet of surface; (3) the protection of the exposed parts of the body by fine gauze nets, especially at night, when alone some species of mosquito make their attacks; (4) the constant use of quinine internally (to the extent of five grains daily) by those who are resident in malarious districts. Estimates have been made in India and elsewhere of the prevalence of the disease—first, by examining the blood of a large number of individuals, and learning the percentage of individuals in whom the parasites can be found (*anæmic index*, Stephens and Christophers); secondly, by ascertaining what percentage of children have enlargement of the spleen.

SLEEPING SICKNESS

(*Negro lethargy. Trypanosomiasis*)

This disease, whose chief feature is indicated by the above names, is endemic in Africa, and is due to infection by a variety of *trypanosoma*, or screw-worm, the *Trypanosoma Gambiense*. This is an elongated wormlike parasite measuring in all from 18μ to 25μ in length, and from 2μ to 2·8μ in breadth; thus it is in length about three times the diameter of a red blood corpuscle. About the middle of its length is a large oval nucleus. At the posterior blunt extremity is a small particle (*centrosome* on *blepharoplast*) from which proceeds a *flagellum*, and this running along the free border of an *undulating membrane* projects beyond the anterior pointed extremity. The parasites are found in the blood, lymph-glands, and cerebro-spinal fluid, and their presence in the latter determines the symptoms characteristic of the disease. In the blood they move slowly in a spiral manner; they are never found in red corpuscles, but are devoured and destroyed by leucocytes. The disease has been produced in monkeys by inoculating them with blood or cerebro-spinal fluid containing the parasites.

Ætiology.—The disease has been known for a long time to occur on the west coast of Africa, between the rivers Senegal and Loanda, and for some thousand miles inland. It has also been recently observed in Uganda, and in other parts of the world; but in these last cases the individuals have always previously resided in Africa. So far it has rarely been observed in others than negroes. It attacks both sexes and all ages, except, perhaps, infants at the breast, or very old persons. The spread of the disease is exactly analogous to that of malaria, the parasite being inoculated into human beings by the bite of a particular variety of tsetse fly, *Glossina palpalis*. Horses and cattle are also liable to trypanosomiasis, with other species of trypanosoma, and of tsetse fly.

Symptoms.—Undoubtedly the trypanosoma may exist in the blood for long periods without producing any symptoms, and possibly sometimes they die out. The first symptoms of trypanosomiasis consist of attacks of febrile reaction lasting a few hours or a few days, separated by intervals of from one to four or more weeks, quick pulse and respiration, languor and debility, pains in the wrists, knees, and ankles, patches of erythema, or congested areas of skin, local and transitory œdemas of the skin of the face or feet, enlarged lymph-glands, and enlarged spleen. In one case death took place after increased fever, rigors, quick pulse, delirium, and Cheyne-Stokes respiration.

When the organism reaches the cerebro-spinal arachnoid cavity, the nervous symptoms develop. The characteristic of the disease is drowsiness, which gradually increases and passes into somnolence, and finally into profound coma. In the early stages, the patient may go about his work, but he is drowsy, listless, languid, or physi-

cally weak; he has a vacant expression, the upper eyelids droop, and the lower lip falls so that the lower teeth are exposed, and the lip is dry and cracked. Later the expression becomes vacant, the face is puffy, and the tongue and hands are tremulous. In a month or two more the prostration is increased; the sufferer walks with difficulty supported by a stick, the lower lip is more everted and saliva dribbles over it. The temperature is raised a little in the evening; and the pulse is often very rapid. In a large number of cases the superficial lymphatic glands are enlarged; and in some cases the salivary glands. Eventually the somnolence is such that the patient lies persistently in one position, either flat on the ground, face downwards, or curled up on one side, or fallen forwards in a kneeling position. During the later stages he eats only what is brought to him; and even stops eating with the food still in his mouth. He would starve if not attended to by others. As he becomes more somnolent, his secretions accumulate about him, and considerable bed-sores form; he may suffer from choreiform spasms, and twitchings; and rapid wasting and diarrhœa occur. Papulo-vesicular and pustular eruptions, and a scurfy condition of the skin have also been noted. In a small number of cases mania develops at some or other period of the illness. The disease lasts from five to fifteen months. It is almost invariably fatal.

Morbid Anatomy.—The chief lesions are found in the nervous system, and consist of chronic meningo encephalitis and meningo-myelitis. The pia-arachnoid is opaque, slightly thickened, and perhaps adherent, and microscopically it is seen to be infiltrated with uninuclear leucocytes, which are also abundant in the peri-vascular spaces. The subarachnoid fluid is increased in amount, of pale straw colour, and slightly turbid, but rarely purulent. The cortex of the brain is deficient in pyramidal cells, and the cord may present a diffuse sclerosis, with degeneration of the axons. The lungs are almost constantly congested and œdematous, and sometimes consolidated in one or more places. The heart is usually flabby and pale. The superficial and deep lymph-glands are much enlarged, and their lymphocytes are much increased. In many organs there is a leucocyte infiltration round the vessels (Low and Mott).

Diagnosis.—This is now determined almost entirely by a microscopical examination of the blood, or of juice drawn from enlarged lymphatic glands, or of the cerebro-spinal fluid drawn by lumbar puncture. In the latter fluid even before the appearance of trypanosomes there will be found numerous lymphocytes, large epithelial cells, and myelocytes, and an increase of serum-globulin and serum-albumin. The enlargement of lymph-glands is a suspicious circumstance.

Treatment.—The drugs which have been most extensively tried are various compounds of arsenic, mercury, and antimony; and especially *atoxyl*, a compound of arsenic and aniline, and the same substance in a different solution, under the name of *soamine*, is

recommended as a subcutaneous injection of a 20 per cent. solution in distilled water or normal saline, warmed to blood heat before use. The daily dose is ·6 cub. cm. for five or six days, ·8 c.c. for another five or six days, and so on by increasing doses till signs of irritation appear, when it may be reduced or stopped for a time, and recommenced later. The results are variable, and patients apparently cured have relapsed and died. In a few also atoxyl has caused blindness. The arsanilate (atoxyl or soamine) is also given in combination with mercury (perchloride subcutaneously), or antimony potassio-tartrate or sodio-tartrate (injected intravenously). Another drug employed apparently with temporary benefit is arsenophenylglycin.

KALA-AZAR

(*Black Fever*)

This is an infective disorder which is prevalent in India, Assam, and China, and has also been found in Tunis, Algiers, Arabia, and Egypt. The disorders known as tropical splenomegaly, Dum-Dum fever, and "cachexial fever" of Bengal are no doubt identical.

The disease is due to parasites, described by Major Leishman and Dr. Donovan, and known as Leishman-Donovan bodies. They are oat-shaped, oval or spherical, about one-third the diameter of a red blood corpuscle (1·5 to 4μ by 1 to 2·5μ), and present two nuclear bodies, one large (*nucleus*, or *macronucleus*), oval and staining rather faintly, the other (*centrosome*, or *micronucleus*) small, rod-shaped, staining deeply, and generally with one end directed to the large nucleus, these two bodies lying against the periphery of the organism opposite to each other. They are found in the spleen, liver, bone-marrow, mesenteric glands, kidney and intestinal ulcers: and they can be obtained from the enlarged spleen by puncture during life. They are well shown by the aid of Romanowsky's stain. They are the early stages of a species of flagellate protozoon (*Herpetomonas*), the further development of which can be observed in suitable media. Thus the parasite multiplies by subdivision, a flagellum shoots out from the neighbourhood of the centrosome, and the organism elongates into a fusiform body, which may reach 24μ in length, having the flagellum and centrosome at one end, while the macronucleus lies near the centre or near the other end.

Various species of herpetomonas have been found in the alimentary canals of flies and bugs, and it appears probable that the bed bug in India is the means of transmission from man to man of the particular variety, *Herpetomonas Donovani*, which causes kala-azar.

Similar organisms (*Herpetomonas infantum*) have been found in Tunis in cases of infantile splenic anæmia which closely resemble kala-azar (Nicolle); and the parasites discovered by Wright in Delhi sore also belong to the same group (*H. Wrighti*).

Symptoms.—The illness often begins with fever, and presents an irregular, remittent or intermittent pyrexia, with anæmia, earthy pallor of the skin, wasting and loss of strength, enlargement of the liver and spleen, occasional hæmorrhages from the nose, gums, or under the skin, pains in the ends of the long bones, and transitory œdema of the face or ankles, and even ascites in cases of much enlargement of the liver; diarrhœa, dysentery, congestion of the lungs or pneumonia are also frequent, and the fatal result is often due to one of these, or to some other accidental complication. The condition of the blood is very constant. The red corpuscles are diminished to 60 or 50 per cent.; the hæmoglobin is diminished, and there is marked leucopenia, so that the leucocytes may be only 2000 per c.mm. The differential count shows great diminution of the polymorphonuclears and a large relative increase of the large uninuclears, which may reach 50 or 60 per cent. of the total leucocyte count.

Morbid Anatomy.—The conditions found after death, in addition to emaciation and muscular wasting, are enlargements of the spleen and liver, dropsical effusions, and ulceration of the large intestine. The spleen is large, firm, deep red in colour, with thickened capsule and trabeculæ; and the parasites are found in the cells of the pulp, and not generally in the Malpighian corpuscles. The liver is dark brown, or mottled brown and yellow, with a moderate degree of cirrhosis. The organisms are found in large endothelial cells, often in the dilated intralobular capillaries. They are never in the hepatic cells, which, however, are atrophied, and show nuclear and fatty degeneration. There are numerous parasites in the uninuclear cells of the bone-marrow, which is converted into the red variety. Ulcers of various sizes are found in the colon and sigmoid, and cicatrices are common.

Diagnosis.—The disease has to be distinguished from malaria and typhoid in the tropics, and possibly from splenic anæmia. The distinctive features are the detection of the Leishman-Donovan bodies in fluid obtained by puncture from the enlarged spleen, and the leucocyte counts where spleen-juice is not available; and negatively the absence of malaria parasites, and of the typhoid agglutinative reaction.

Prognosis.—The disease lasts several months, and has been fatal in more than 90 per cent. of the cases.

Treatment.—Opinions differ as to the value of quinine: it is said by some to be valuable as a prophylactic, while others regard it as useless; but Rogers asserts that cases have certainly recovered under daily doses of 60 or 90 grains given over considerable periods. Atoxyl and arsenophenylglucin have been used for the allied cases in Tunis.

DENGUE

Dengue or Dandy Fever is a disease occurring only in or near the tropics. It begins with fever of short duration, accompanied by pains in the joints and limbs; then, after a short interval of apyrexia, there is a relapse or second fever, and often a cutaneous rash. Second and third relapses even occur.

Epidemics of this disease have been observed in India, Burmah, Persia, in Egypt and other parts of Africa, in North and South America, and the West India Islands. In its short period of *incubation* (from one to three days), in the suddenness with which its epidemics burst upon a community, and in its conveyance, apparently by human intercourse, along trade routes, it closely resembles influenza. Of recent years, however, transmission by the bites of mosquitoes has been suspected.

Symptoms.—The patient is suddenly seized with pain in one or other joint, often in the finger, with headache and fever. Other joints are afterwards affected, and the pains shift about, lessening in one part as they appear in another, and affecting the muscles as well as the joints. The headache is accompanied by pains in the eyeballs. The temperature rises to 102°, 103°, or even 105°, and the pulse is commonly a little over 100. In many cases also, this first fever is accompanied by a rash, either redness of the face or a general red colour; and the throat may be sore. But the rash disappears in twenty-four hours, and about the same time the pyrexia terminates—sometimes suddenly with critical symptoms, such as sweating. The pains abate, and the patient is in comparative comfort, but weak for two, three or four days; when again he becomes feverish and a rash appears, which is either diffused like scarlatina, or maculated like measles, or elevated like lichen tropicus, or, it may be, resembles urticaria. It generally causes some itching. It begins on the palms of the hands, and spreads to the whole of the body. It lasts from a few hours to two or three days, and is followed by desquamation. In this second fever joint pains again occur, and may persist or relapse after the subsidence of the fever. The whole duration of the disease is about eight days, unless where second relapses follow, or where the joints remain swollen, painful, and deformed, as they may do for months afterwards, even becoming partially ankylosed. It is, as a rule, only fatal to infants and old people. Mild forms may present only malaise, sore throat, and the second or *terminal* eruption. In severer forms there may be coma, hyperpyrexia, failure of the heart, œdema of the lung, or cyanosis.

Little is known of the **Pathology** of the disease. From the rash and joint pains it has been called *scarlatina rheumatica*, but it presents interesting points of resemblance to relapsing fever.

Treatment.—After attention to the bowels, salines and diaphoretics are recommended for the general condition, and quinine

has been said to shorten the paroxysms. For the joint pains tincture of belladonna, in doses of 10 or 15 minims, is much recommended, and morphia or Dover's powder may be given. Heat of skin may be relieved by cold sponging, and the irritation of the second rash by the use of camphorated oil. During convalescence iron and quinine should be given.

BERI-BERI

(*Kakke*)

This is an endemic disease, consisting essentially of a multiple peripheral neuritis, which causes paralysis and anæsthesia, especially of the lower extremities, with cardiac dilatation and dropsy in varying degrees.

Ætiology.—The important feature in its causation is its relation to locality. It has been observed chiefly in Japan, China, the Malay Peninsula, and slightly in India and Ceylon; in some parts of the west coast of Africa; in the West India Islands; and the eastern parts of South America. It is, however, frequently conveyed to European ports. The most susceptible time of life is between 20 and 30, and males are more liable than females. The disease occurs in outbreaks, is frequent in towns and inhabited places, occurs on board ship, and is apparently promoted by overcrowding; but it does not seem to be transmitted directly from man to man, nor to be conveyed by water.

For a long time it has been suspected that food was responsible for this disease; and instances have now occurred in which the disease has broken out in parties of coolies fed on white or uncooked rice, while other parties fed on steamed or parboiled rice have been entirely exempt. So far, however, no organism, bacillary or protozoal, has been discovered; and the explanation of the above facts appears to be that parboiling renders the grain tougher, and prevents the removal of the pericarp which is effected by milling in the case of white rice. The pericarp contains a large amount of phosphorus, the loss of which, to those who live on white rice, is the cause of nerve degeneration in this disease. This view is supported by experiments on fowls (Fraser and Stanton).

Symptoms.—These are, for the most part, the weakness or paralysis of muscles, muscular atrophy, and sensory disturbances, which are characteristic of multiple neuritis (*see* Diseases of the Nerves); but there are added in this form certain features not commonly seen in the familiar cases of neuritis due to alcohol, diphtheria, or plumbism—namely, œdema of the legs, or even extensive anasarca, and severe or fatal dyspnœa from cardiac failure or œdema of the lungs.

There is, however, considerable variety in the effects produced by these lesions: in some anasarca is pronounced or extreme (*wet beri-beri*); in others, the muscular atrophy is the chief feature, and

the patients are thin and emaciated (*dry beri-beri*); and intermediate conditions occur.

The disease often begins slowly with a state of languor, with weakness of the legs and knees, pains in the calves, and slight dypsnœa; or the occurrence of weakness and numbness of the legs, and pains in the calves may be almost sudden. As the disease develops, there are loss of power and muscular atrophy beginning in the extensors on the front of the leg, and then affecting the other muscles of the leg and thigh, as well as later, the extensors of the hand, the biceps, and it may be, the abdominal muscles, the diaphragm, and the intercostals. The knee-jerk is generally soon lost. When the legs are chiefly affected, the patient has a gait characteristic of foot-drop: the heel is lifted high to clear the ground, and the toes came down before the heel. Anæsthesia is only noticed in the skin over the tibiæ, and may extend to other parts of the limbs and trunk. Hyperæsthesia of the muscles, especially of the calves, tenderness of nerve-trunks, and painful cramps occur, as they do so often in multiple neuritis from other causes. A little œdema over the shins appears to be almost invariable. When the anasarca is extreme the urine is scanty. There are in most cases palpitation and dyspnœa, and bruits are heard at one or more of the cardiac orifices. The pulse is soft and rapid. The temperature is generally normal or even subnormal.

Cases often last about five weeks; some a shorter time, others as much as twelve months or more. The danger lies in cardiac failure, with rapid irregular action, or in asphyxia from œdema of the lungs; and these conditions sometimes arise quite suddenly and carry off the patient. Sometimes fatal exhaustion follows vomiting.

Morbid Anatomy.—*Post mortem*, in addition to the œdema and anasarca seen during life, there are ecchymoses under the serous membranes, in the muscles, and in the sheath of the nerves; the lungs are engorged and œdematous, the right side of the heart is dilated, and its muscular fibres may be degenerated. The mucous membrane of the pyloric end of the stomach, and of the duodenum, is congested or presents punctiform hæmorrhages (H. Wright). The muscles are either atrophied or swollen, and the nerves show evidence of inflammation or neuritis.

Treatment.—There is no specific for this disease. Early cases apparently due to uncooked rice have sometimes recovered with change of food: and change of locality was recommended so long as it appeared likely that the poison was in the soil or air. Otherwise little can be done. In late stages iron and strychnine internally, and faradisation and massage to the limbs are recommended. For serious cardiac failure active purgatives, full doses of nitroglycerine or nitrite of amyl, and, if necessary, bleeding should be employed.

YELLOW FEVER

Yellow fever is an acute specific disease, occurring within certain geographical limits, and characterised by fever of short duration, a yellow tint of skin, severe gastro-intestinal disturbance, and albuminuria, or suppression of urine.

Ætiology.—This disease was first met with in 1647 in the West Indies, and is peculiar to that group of islands, to North and South America, and to the west coast of Africa. It has, indeed, been occasionally carried to other parts of the world—for instance, in 1865 to Swansea, where a slight epidemic was the result; but it has never maintained itself away from the localities mentioned, which lie between the latitudes of 48° north and 35° south. It occurs almost exclusively in crowded towns, and especially in those having a maritime commerce; and for the most part it is confined to low levels, and is rarely found higher than 2000 feet above sea level; but epidemics have occurred in the Andes at an elevation of 11,000 and 14,000 feet.

It requires a high temperature, not less than 70° F. or 72° F., according to most writers, but it has been known to occur at lower temperatures, *e.g.*, 65°. It is stated to be stopped absolutely by cold sufficient to freeze the earth. It is most fatal in the summer months—from May to August.

It affects all ages and both sexes; but it does not commonly attack the same individual a second time. The supposed smaller susceptibility of negroes than of white men is attributed by Sir R. Boyce, not to racial differences, but to the immunity which an indigenous people may acquire by long contact with an endemic disease.

Abundant evidence has now been afforded that the infecting agent of yellow fever is transmitted from man to man by means of a species of mosquito, the Tiger or Brindled Mosquito (*Stegomyia fasciata*), in the same way as the organisms of malaria and sleeping sickness. The mosquito dwells in towns, and not in marshes or swamps; and it breeds in clear water receptacles in the yards of houses, in cisterns, barrels, and tins used for the storage of water, in old bottles, meat and milk tins, flower pots, &c., and in water collected in canoes.

Though Sanarelli found a bacillus (*B. icterodes*) in the blood and tissues, but not in the alimentary canal, and Durham and Myers isolated a minute bacillus from several organs, as well as the intestinal contents, the identity of the organism has not been established.

Symptoms and Course.—The period of *incubation* is five days: that is to say, the symptoms appear approximately five days after the person has been bitten by an infected mosquito.

Sometimes suddenly, sometimes after a short period of languor, headache, or malaise, there are chills or rigors of more or less severity. These are followed by febrile reaction, the temperature

rising in two or three days to 105°, or even higher. There are generally frontal headache and severe lumbar pains or pains in the joints. The pulse varies from 100 to 120, mostly not so quick in proportion to the temperature as in some other fevers. The tongue is generally covered with a thick creamy fur, leaving the edges and tip bright red. There are mostly tenderness and pain in the epigastrium, with nausea or vomiting. About the second or third day the conjunctivæ become yellow, and jaundice spreads to the whole body. The urine is scanty, with diminished urea and uric acid; and it constantly contains albumin, which may be found as early as the second day of the illness. Bile-pigment appears a few days later.

On the fourth day there is often a remission of temperature, and the general pains subside. This may be the commencement of convalescence, the yellow tint gradually clearing up, albumin disappearing from the urine, and the patient recovering in two or three weeks.

But in many cases the more serious symptoms continue. The patient may fall into a fatal collapse; or the temperature may rise again for a few days, though not to the same height as before. The jaundice deepens and petechiæ appear under the skin; the urine is still less in amount, while the albumin increases, and casts are present; and finally, complete suppression for days may occur. The vomiting becomes frequent. At first only the gastric contents, mixed with more or less bile, are discharged, but afterwards occurs the so-called "black vomit" due to the presence of blood, often in a form which is likened to coffee-grounds. This is sometimes preceded by a limpid, ropy, opalescent fluid (white vomit). When blood is discharged by the stomach it is generally passed by the motions as well. Hæmorrhage may take place also from the nose, mouth, or gums; and the tongue by this time has lost its fur, and becomes dark brown, raw, and covered with blood-crusts or sordes. Delirium becomes pronounced, or the patient may sink into coma. The mortality varies from 5 to 75 per cent. in different epidemics. Death takes place sometimes within a few hours of the onset, more often after the remission of fever, either from collapse, from profuse hæmorrhage, from coma, which is generally attributed to suppression of urine and uræmia, or from typhoid or adynamic conditions. The temperature sometimes rises to 108° to 110° immediately after death.

Anatomical Changes.—The changes described are anæmia and acute fatty degeneration of the liver; acute catarrh of the stomach, with ecchymosis, or hæmorrhagic erosions; hæmorrhages in the tissue of the lungs and under the pleuræ; pale, yellow-brown colour of the muscular substance of the heart, or acute fatty degeneration or ecchymoses; and acute glomerular and parenchymatous inflammation of the kidneys, sometimes with miliary abscesses. The spleen differs strikingly from that of malarial disease, in being usually quite unaffected. The blood contains an excess of urea, which may reach 4 per cent.

Diagnosis.—There is generally little difficulty about the diagnosis in localities where the disease is prevalent. The early acute symptoms may present a certain resemblance to the onset of such illnesses as small-pox or pneumonia. Later, it has mainly to be distinguished from malarial fevers. These last are endemic, and not transmissible so as to affect healthy persons in new localities: they present intermissions of actual health, or, at least, remissions between the exacerbations, which recur with regularity: the spleen is enlarged, and the individual is not protected by his illness from future attacks.

Relapsing fever may be accompanied with jaundice, but the primary fever is longer, and the interval is one of very great improvement: the spleen is enlarged. Acute yellow atrophy of the liver begins more gradually, often with an apparently simple jaundice.

Prognosis.—The unfavourable signs are a very high temperature, abundant albuminuria, suppression of urine, black vomit, or pronounced nervous symptoms.

Treatment.—An important distinction from malarial diseases is that neither quinine nor any other drug has a similar influence over yellow fever. Quinine is given, but chiefly as a tonic, after the more acute stages are over. The treatment usually employed is to give a laxative or enema to clear the bowels, and then to make use of salines and diaphoretics, and relieve symptoms as they arise. High fever may be met by cold sponging; vomiting by ice internally, by very small doses of morphia or chlorodyne, or by lime-water; the action of the kidneys may be stimulated by warm baths or vapour baths. The diet must be fluid but abundant, and alcohol will be required; but writers recommend that it shall be given somewhat dilute, either as brandy with much water, or as champagne. Sternberg recommends the following as being of great service in checking gastric irritability, increasing the amount of the urine, and giving favourable results in respect to recoveries: one-third of a grain of perchloride of mercury and 150 grains of bicarbonate of sodium in 2 pints of pure water, of which 3 tablespoonfuls are to be given every hour, ice-cold. Sanarelli has prepared from horses a curative serum which is said to be of value; but being bactericidal and not antitoxic, it must be used in the first two or three days of the illness.

Prevention.—This should be carried out by the same methods as are employed for malaria, namely, the protection of the individual from the bites of mosquitoes, and the extermination of these insects by all available means (*see* p. 101), including fumigation of dwelling rooms by burning formalin, sulphur, pyrethrum, or a mixture of camphor and carbolic acid. Complete success has attended these measures in Havana, New Orleans, Panama, Rio, and some other infected localities. Notification is, of course, necessary, and quarantine for five days after infection of those who are not immune is also recommended.

A yellow fever patient can infect mosquitoes during the first

three days of his illness; and a mosquito so infected can, after the lapse of about ten days, transmit the disease to healthy persons. It retains this power of transmitting the disease for many weeks.

PELLAGRA

Pellagra (*pelle*, skin, and *agro*, rough; or *pelle* and *agra*, as in podagra) is a disease occurring in Spain, Italy, and Southern Europe, of which the chief features are an eruption on the skin, and symptoms of mental depression ending in dementia. For years it has been supposed to be due to some disease in the maize, or Indian corn, upon which the peasants of these countries feed; and the frequent presence of *Aspergillus fumigatus* in such diseased corn seemed to supply the information as to the actual cause. Recently, however, in March 1910, Dr. Sambon, studying the disease in Northern Italy, in the provinces of Bergamo, Brescia, Milan, and Padua, satisfied himself that maize had nothing to do with it; but he found that the centres of the disease corresponded to the neighbourhoods of streams infested by the larvæ of certain *Simuliidæ* or sand-flies. The sand-flies remain in the neighbourhood of the streams. They bite in the early morning, and at sunset, and never in the hottest part of the day. If Dr. Sambon's observations be confirmed, pellagra must be regarded as related in its ætiology to malaria, yellow fever, and sleeping sickness.

Ætiology.—The disease has been known chiefly in Spain, Northern Italy, France, and Rumania, and more recently in Hungary, Corfu, Egypt, and the West Indies. It occurs at all ages, but is uncommon in infancy and in old age: it attacks the sexes equally. The subjects are mostly peasants of the poorer class, dwellers in the country, and in Egypt the fellaheen mostly, as well as masons' labourers, brickmakers, potters, pedlars, readers of the Koran, and beggars (Sandwith). The disease begins commonly in the spring of the year; that is, in Europe about March, April, or May, but in Egypt its first appearance is generally in January or February. It is in the spring that the larvæ of the sand-fly assume the winged condition.

Symptoms.—The early symptoms are bodily weakness, headache, sleeplessness, and depression, vague pains and cramps, vertigo and dyspepsia. Then, as a rule, appears the *eruption*, which has caused this disease for a long time to be classed as a skin disease. This is an erythema, like a severe sunburn, occupying the face, the front of the chest, the hands, forearms and elbows, the feet, insteps, legs and knees. These are the parts most exposed to the sun, and if a greater or less area is exposed there is generally a corresponding difference in the area of the erythema. The extensor surfaces of the limbs are more affected than the flexor. With the redness there are itching and burning sensations; and petechiæ and bullæ may occur. After about a fortnight the redness subsides, and the epidermis is shed

in gray or brown flakes, leaving a surface thickened and often striated. After repeated attacks in successive years, the skin remains wrinkled, dry, and atrophic.

The disturbances of the *digestive system* may be pronounced, in the form of epigastric pain and tenderness, flatulence, and thirst. The tongue is bared of epithelium, and in late stages diarrhœa almost always sets in.

The third feature in this disease is the occurrence of *spinal* and *mental* symptoms. The former consist of pains in the back, with tenderness of the dorsal or lumbar spines, increase of the knee-jerk in 75 per cent. of the cases, weakness and stiffness of gait, or tremor. In quite late stages there may be definite paralysis, with loss of knee-jerks, and of vesical and rectal control. On the mental side are observed from the first depression, gloom, or stupor, insomnia and restlessness. And with repeated recurrence of the attacks for three or four years, the patient becomes melancholic, with occasional attacks of mania, and ultimately lapses into a hopeless state of dementia. Pellagrins contribute a large proportion of lunatics to the asylums of the countries in which this disease occurs, and, as bearing on its ætiology, Sandwith observes that such chronic lunatics may develop a new erythema every spring in spite of the fact that maize is never given to them as food.

Morbid Anatomy.—The most marked changes are found in the spinal cord, consisting of sclerosis or degeneration of the posterior columns and of the lateral columns. The former are most affected in the cervical and upper dorsal regions, the latter in the middle or lower dorsal regions. The brain is wasted generally. There is pigmentation of the anterior cornua of the spinal cord, of the solid viscera, and of the skin: while fatty degeneration of the heart, liver, and kidneys, and fibrosis of the liver, kidneys, and spleen are common.

Diagnosis.—This is generally easy in the countries where the disease is endemic. Some confusion with other nervous diseases, such as tabes, may occur if the eruption is absent, as it sometimes is.

Prognosis.—This is essentially bad; but early cases seem to have been improved, or even cured.

Treatment.—Hitherto this has consisted in abstention from the supposed cause, maize, as a food, and in the use of perfect hygienic measures, as regards food, fresh air, cleanliness, &c. No drugs have any influence other than general tonics, such as strychnia and arsenic and cod-liver oil. Sandwith has given fresh bone-marrow.

DYSENTERY

This is an inflammation of the large intestine or colitis, and sometimes of the lower part of the ileum, resulting in extensive ulceration, and accompanied by fæcal discharges which consist almost entirely of mucus, muco-pus, or blood. In some forms a

protozoon, the *amœba dysenteriæ*, is found abundantly in the stools (amœbic dysentery); in other cases a bacillus, *B. dysenteriæ*, is found (bacillary dysentery).

The amœba of dysentery (*amœba dysenteriæ, entamœba histolytica*) is from three to five times the diameter of a red corpuscle, and occurs not only in the stools but in the wall of the diseased bowel, in the associated hepatic and pulmonary abscesses, and in the sputa.

The bacillus (*B. dysenteriæ*) is a flagellated bacillus holding a position intermediate between the B. typhosus and the B. coli communis. Various strains are known to occur (Shiga's I. and II., Flexner's, Kruse's, Vaillard's). It is agglutinated by the serum of patients suffering from bacillary dysentery, but not by that from a case of amœbic dysentery.

Ætiology.—Dysentery is especially a disease of the tropics, where it is endemic in certain regions, but also spreads in an epidemic form. It is within the limits of 35° or 40° north and south of the equator that it is especially frequent. It is not climate alone, however, that determines its occurrence, but rather terrestrial conditions, since many places are exempt from it, and those that are favourable to it are often also the homes of the severer forms of intermittent and remittent fevers. It is generally most prevalent during the rainy season. Its invasion of any individual is favoured by depressing conditions, such as exposure to cold, bad hygienic surroundings, a preceding malarial attack or enteric fever, alcoholic indulgence, or ingestion of unsuitable food, rotten fruit, &c. In temperate climates epidemic dysentery results from overcrowding and insanitary conditions, and occurs in military camps; thus it raged during the Franco-German war of 1870. In the South African war of 1899 to 1902 it attacked thousands, and its spread was attributable to polluted water, and to infection of food from the fæcally-impregnated soil by means of flies and dust storms. It has also broken out in prisons under conditions probably dependent on impure supply of water or air. It is even now not uncommon in asylums for the insane (*asylum dysentery*), where the hygienic conditions may be good; and its invasion is thought by some to be due to the lowered nervous force of the sufferers, and by others to unfavourable local conditions, such as habitual constipation.

Sporadic cases of dysentery arise from unsuitable food, bad meat or unripe fruit, or from drinking water impregnated with organic matter. It is probably contagious only, in the same way as cholera and typhoid fever, through the medium of the dejecta, by which either the food or the drinking water is contaminated.

Of the two forms of dysentery, both occur in India and in the Philippine Islands; amœbic dysentery occurs in the United States; the bacterial variety in Japan. Bacteria have been found in sporadic acute dysentery occurring in the British Isles, and in

asylum dysentery; and amœbæ were not found in the dysentery of the South African war.

Symptoms.—In cases where it has been possible to fix the period of *incubation*, this has varied from a few days up to eleven.

The patient is first taken with diarrhœa, and passes daily from two to five or six yellow or brownish-yellow loose fluid motions. There is some abdominal pain, and a certain amount of malaise, with loss of appetite. After three or four days the stools become suddenly more frequent, so as to reach ten, twenty, forty, or sixty or more in the twenty-four hours, and they acquire the special characters of *dysenteric stools*. Each discharge is very scanty, and may not be more than a few drachms; but the total quantity in twenty-four hours may be from thirty to fifty ounces. The stools at the height of the disease contain no natural fæcal matter, but consist entirely of mucus, serum, blood, and pus, in varying proportions, with detritus, and perhaps shreds of necrosed mucous membrane. In the early stages the discharges consist of yellowish transparent mucus, with small lumps or streaks of blood; in later stages blood is more abundant, in clots or lumps, floating in a red serous fluid. Sometimes pure blood is passed, either from early congestion of the mucous membrane, or from ulceration of the walls of the vessels; pus may be present alone, or there may be a blackish or brownish-red offensive slimy fluid, containing portions of tissue that have sloughed away. Sometimes the stools contain curious tough masses of mucus, like frog's spawn, or boiled sago, which Heubner thinks have been formed by the coagulation of mucus in some of the ulcerative depressions of the diseased mucous membrane. The odour of the motions in the advanced stages of the disease is thought by some to be peculiar to dysentery. Two other frequent symptoms are *tormina*, or griping abdominal pains, and *tenesmus*, or the painful straining or desire to go to stool. The latter only occurs when the lower end of the rectum is affected, and it is accompanied by burning pains in the rectum and anus. It often results in no evacuation, or at most in a very small quantity. Frequent micturition or strangury is sometimes associated with it. These severe local conditions soon react upon the general health. The patient rapidly loses flesh and strength, the face is pale, sallow, or tinged with yellow, the tongue is covered with a thin fur, there is moderate fever, the temperature rising to 101° or 102°, with headache, dizziness, loss of appetite, and much thirst. In milder cases the symptoms abate after eight or ten days; the pain and tenesmus are less, and the stools gradually acquire more consistence, become more feculent, and are finally quite natural. In the severer cases, the discharges are more and more mixed with blood and pus, or become greenish-black and offensive; while the patient's exhaustion increases, the motions pass uncontrolled, the anus and surrounding parts are excoriated, and death is preceded by collapse, with pinched features, livid extremities, hoarse voice, and scarcely perceptible pulse. Vomiting, which is an early symptom in most cases,

becomes severe and continuous in the graver forms. The urine is scanty, wanting in chlorides, but not commonly albuminous.

Other cases pass into a condition of *chronic dysentery:* the stools are sometimes almost natural, at others consist of varying mixtures of mucus, pus, and blood, which have the same offensive odour as in the acute stages; and this may continue with more or less severity for months or years. The patient remains thin and weak, but may with judicious treatment recover, or may die eventually of exhaustion, or as a result of such complications as hepatic abscess, peritonitis from perforation of the bowel, or stricture from contraction of the cicatrix.

The features by which amœbic dysentery is distinguished from other forms appear to be a slow onset, a subacute or chronic course, the frequency of relapses, the occurrence of hepatic abscess, and the absence of the serum reaction (*see* pp. 19, 52).

It is difficult logically to exclude cases described hereafter as *ulcerative colitis* from the category of dysentery. If organisms have not been found, it may be from investigation at too late a stage.

Complications.—One of the most important complications is *abscess of the liver*. This is generally a single extensive lesion, the so-called *tropical* abscess: less frequently there are numerous small abscesses. The former is the characteristic of amœbic dysentery, and is produced either by infection through the portal vein, or, possibly, by direct infection from the adjacent colon. Multiple abscesses are probably always due to portal infection, and occur in bacterial dysentery. *Peritonitis* sometimes results from perforation of a dysenteric ulcer. Dysentery is sometimes followed by a multiple *arthritis*, lasting a few weeks, with slight swelling and tenderness of the joints, and moderate fever.

Pathological Anatomy.—Dysenteric inflammation of the intestines occurs in every degree of intensity, involving in nearly all cases ulcerative processes, and in many gangrene or sloughing. Authors have accordingly described different forms, as the *catarrhal* and *diphtheritic* of Heubner, and the *ulcerating* and *gangrenous* of Kelsch and Kiener; but the disease is essentially the same in all. In the *catarrhal* or simply *ulcerative* forms, the mucous membrane is intensely injected, reddened and swollen; the redness is often confined to the prominent edges of ridges of mucous membrane, or it may be irregularly distributed; and the surface is covered with mucus tinged with blood. The solitary follicles become swollen comparatively early, and after a time the surface of the follicle is abraded, and a little pit results. The mucous membrane then becomes infiltrated with pus, and the submucous and muscular tissues are swollen. Finally the softened mucous membrane breaks down, and ulcers of irregular shape and varying extent are formed, which often at first do not involve the solitary follicles, but leave them surrounded by a little ring of mucous membrane. Ultimately these also may be separated and shed. The bowel then presents a red or yellowish-brown colour corresponding to

the ulcerated parts, with the patches or islands of bluish-red or gray colour, representing the inflamed mucous membrane, still persisting.

When *gangrene* occurs, patches of iron-gray, brownish-red, dark red, or black colour are met with, which evolve a gangrenous odour. Such patches may be quite small, or some inches in diameter. When cut into, they yield a sanious liquid, mixed perhaps with pus; and they are found to involve the submucous tissue extensively, and in some cases the muscular coat, and even the peritoneal covering. The bowel thus affected often contains a brownish liquid of gangrenous odour, sometimes mixed with blood. When the sloughs separate they leave ulcers with undermined edges, which in favourable cases cicatrise. The mucous membrane between the sloughs or ulcers is hyperæmic and infiltrated with serum and blood. The conversion of the mucous membrane and submucosa into a rigid homogeneous tissue as an acute process previous to gangrene (Heubner's *diphtheritic* form) is said by Dr. Davidson to be only of occasional occurrence in the tropics.

In dysentery associated with amœbæ and occurring at Baltimore, the lesions were œdematous thickening of all the coats of the intestines, especially of the submucosa, gelatinous softening and suppuration of the latter, and formation and extension of ulcers by sloughing of the mucosa over it. (Councilman and Lafleur.) The formation of black sloughs is not common. (L. Rogers.)

The parts of the bowel liable to be diseased in dysentery are the rectum, colon, and cæcum; and the disease extends in some cases beyond the ileo-cæcal valve to the ileum. The disease is often advanced at one part, while still only commencing in another. In amœbic dysentery the lesions are often confined to the upper half of the colon. Catarrhal conditions are curable, and even from diphtheritic or gangrenous stages recovery is possible if the lesions are not too extensive. Granulations spring up over the ulcers, and cicatrices result, which are at first below the level of the islets of retained mucous membrane; subsequently the surface becomes more uniform. After extensive sloughing, the cicatrices may present bands, or cords, or contractions, which later may interfere with the passage of the fæces. Besides the changes in the lower bowel, there may be some catarrh of the stomach and small intestines. The mesenteric glands are swollen and reddened. The liver is swollen and hyperæmic; the spleen, as a rule, is small. The body generally is anæmic and wasted. Persistent suppuration of the dysenteric ulcers and of the sinuses left by submucous abscesses is the cause of *chronic dysentery*.

Diagnosis.—This is determined by the character of the stools, with the associated tenesmus and griping; but the two latter symptoms may be absent. Local diseases of the rectum alone may give rise to somewhat similar conditions. Patients with *cancer of the rectum* have a good deal of straining, and pass mucus streaked or tinged with blood; they are mostly elderly people, and a rectal

examination quickly decides the question. The passage of blood in *intussusception* may also mislead. A simply overloaded rectum may give rise to frequent evacuations of a little mucus; but more liquid discharges of reddish serous fluid, containing lumps of mucus, with blood and pus, are characteristic of dysentery. On the other hand, dysentery seems sometimes to present so few symptoms as to be scarcely appreciable even to the patient. Lesions of the lower end of the bowel may be seen with the sigmoidoscope.

A more complete diagnosis will be made by searching the stools for organisms. Amœbæ are best found by examining on a warmed slide a piece of bloody mucus, or slough taken from fæces collected in a warmed bed-pan, and free from urine: the amœbæ may be then living and active (Sandwith). In the bacillary form the bacilli may be found in the stools, or more easily in fluid gently scraped from the mucous membrane of the rectum. In this form also, a diagnosis may be sometimes established by the agglutinative action of the patient's serum on Shiga's bacillus.

Prognosis.—The mortality in different epidemics has ranged between 30 and 80 per cent. Gangrenous stools, free bleeding, severe vomiting, and indications of collapse are unfavourable; and the disease is specially fatal to infants, old people, sickly individuals, and habitual drinkers.

Treatment.—The patient should be kept warm in bed, and should use the bed-pan for all his evacuations. He should have a light fluid diet, consisting of milk, milk and lime-water, chicken broth, sago, arrowroot, or tapioca given warm, as cold liquids are liable to stimulate the peristaltic movements of the bowel.

In regard to therapeutic measures, the treatment of the two forms is somewhat different. In the *bacillary* cases a laxative method has been considered best, by means of castor oil, calomel, sodium sulphate, or magnesium sulphate. The last two are more commonly employed, given in drachm doses every hour until the motions become copious, feculent, and free from blood and mucus, the temperature has fallen, and pain and tenesmus have ceased. The salts require to be given in different cases for one or two days, occasionally three, but rarely more. Bismuth and opium are of use afterwards.

If pain and tenesmus are acute an opium or morphia suppository may be given, or a small saline enema. Abdominal pain may also be relieved by poultices. Stimulants should only be given in small doses, except in cases of collapse. As the case improves, solid diet of tender meat and farinaceous food, but not vegetables, may be allowed; and iron and bitter tonics are useful.

Several anti-dysenteric sera have been employed in the last few years, which possess both bactericidal and antitoxic powers (Shiga, Kruse, Dopten and Vaillard, and others): and the mortality of the disease is believed to have been much reduced. They must be given in the early days of the disease. The use of vaccines obtained from the bacillus found in the case, associated with opsonic observations, has also been advocated.

In long-standing cases enemata may be used similar to those used more frequently in amœbic dysentery.

If amœbic dysentery begins acutely, its treatment may be begun in the same way as in the bacillary form; that is, by saline laxatives. But it is especially in this form that ipecacuanha has been found to be such a valuable remedy. The ipecacuanha should be given in doses of 20 to 30 (or even 60) grains, suspended in 2 drachms of syrup of orange peel and half an ounce of water. Entirely to obviate its emetic action a small dose of tinctura opii or chlorodyne may be given half an hour before, and the patient should be kept lying down afterwards, and nothing should be given to drink for some time; at most a little ice may be sucked. In many cases nausea occurs; but vomiting is exceptional, and then only after an hour or two, so that the ipecacuanha is probably retained. A similar dose may be given again after eight or ten hours, or if either is rejected, it may be repeated as soon as the stomach is again quiet. Doses of ten or twenty grains may then be given night and morning; or the larger doses may be continued twice or three times a day if the symptoms are severe. The tenesmus and tormina are sometimes at once checked or diminished, the stools soon become feculent and natural, and the mucus and blood disappear. When this stage is reached, the ipecacuanha may be discontinued, and astringents, such as bismuth and opium, may complete the cure.

Chronic dysentery is also benefited by ipecacuanha. The milder cases, which are occasionally seen in sailors landing in London, are often quickly cured by doses of 5 or 10 grains, given twice or thrice daily. In obstinate cases benefit is derived from astringent or antiseptic injections into the lower bowel. The substances most commonly used are boric acid (a drachm to the pint of warm water), nitrate of silver (10 or 15 grains to the pint), and, especially in amœbic cases, quinine (10 grains to the pint). The quantity injected should be at least 30 ounces, and, if possible, two or three pints: and the fluid should be allowed to run slowly into the bowel from the funnel or reservoir through a long soft rubber tube introduced some distance into the rectum.

A warm climate, continued rest, a carefully regulated diet, and tonics, such as iron to counteract anæmia, are also desirable.

Prevention.—The methods adopted must be similar to those practised in the case of cholera and enteric fever. If dysentery has broken out in any place, every insanitary condition, such as uncleanliness, imperfect sewage arrangements, contaminated water-supply, &c., must be dealt with. Stools should be disinfected, as well as linen, instruments, and utensils. Overcrowding should be avoided. Individually every one should be very particular about his diet, avoiding unripe fruit, indigestible food of all kinds, or those that are likely to lead to constipation; and he should carefully guard against chills by adequate clothing.

DIPHTHERIA

Diphtheria (from διφθέρα, a prepared hide, piece of leather) is an acute infectious disease, of which the essential clinical feature is a peculiar inflammation of surface tissues resulting in the formation of a so-called "membrane." This commonly affects the mucous membrane of the mouth, pharynx, nose, or larynx; more rarely some other mucous membrane (conjunctiva, vagina), or the abraded skin, or the surface of a wound.

The specific micro-organism of diphtheria is the bacillus described by Klebs and Loeffler. It is a short rod, but varies in length in different circumstances, from to 2·5μ to 6μ, so that short, medium, and long varieties have been described. It is slightly curved, and often clubbed at one end: it is non-motile, and does not form spores. It stains well with aniline dies, by Gram's method, and with Loeffler's methylene blue. The *B. diphtheriæ* is found for the most part in the deeper layers of the diphtherial membrane: but may be present in small numbers in the lymph-glands, and in the liver, spleen, and kidneys. In the throat it may be associated with other organisms, such as streptococci and staphylococci.

Ætiology.—Diphtheria is contagious, being conveyed through the atmosphere immediately surrounding the patient, as well as by clothes and other objects, and instruments used in the surgical treatment of cases. It can also, like scarlatina and typhoid fever, be distributed with the milk-supply, and probably in such a case it may originate, like scarlatina, in a disease of the cow's udder. It can be communicated to cats, and conveyed again by them to children. Its power of infection is, as a rule, not so great as that of scarlatina and small-pox, or, at any rate, its diffusion in the air seems to weaken it, so that those who catch the disease are generally those who have been in close contact with the patient. On the other hand, there is evidence to show that it may be sometimes transmitted over considerable distances of country by the wind. In spite of the facts which suggest that it may arise in other ways —for instance, its origin in connection with bad drains, and in newly inhabited houses which have been built on "made soils," and the occasional prevalence of ordinary sore throats just before an epidemic of actual diphtheria—it is probable that infection from a preceding case is the source of it in all instances. An occasional source of contagion is the *diphtheria-carrier, i.e.*, the person who has had the disease months previously, who is free from all symptoms, but from whose throat or nasal passages the Klebs-Loeffler bacillus can still be cultivated (*see* p. 42). Besides the conditions already noted as affecting its spread, we may observe that it sometimes complicates measles and scarlatina (*post-scarlatinal diphtheria*), and that it is more frequent in rural than in urban districts, especially in the more exposed parts of the former. It affects both sexes and all ages, but it is especially frequent in children up to ten or twelve years of age.

Symptoms and Course.—The *incubation* lasts from two days to five or six days. The disease, though febrile, does not often begin in the acute way characteristic of small-pox, scarlatina, and others; there are generally malaise, loss of appetite, and headache, and there may be nausea, vomiting, or shivering. Sore throat is soon complained of, and if the throat be examined one or both tonsils, or the palate and uvula, are seen to be reddened and swollen. Within a short time one or more patches of a creamy white deposit form on the inflamed surface. Such patches may form simultaneously on both tonsils, or on one before the other, or they may come on the uvula or the arch of the soft palate; and it is on these parts more than any other part of the mouth that they most frequently appear first. On the soft palate it can generally be seen that the patch is surrounded by an areola of deep red mucous membrane. If the "membrane" is stripped off, a raw surface is left, bleeding from a few points; and within a few hours another patch forms. In some cases the membrane extends on to the fauces, and forward on to the hard palate, presenting a continuous dense layer of yellowish-white or wash-leather colour. Coincidently with the inflammation of the throat the lymphatic glands at the angle of the jaw enlarge, and they can always be felt on one or both sides, according to the lesions within. Sometimes the typical membrane is preceded by a gray mucous secretion. Gangrene occurs occasionally in the severe cases.

The temperature of diphtheria is very variable, and runs no definite course; it may rise to 103°, 104°, or 105°, but is often throughout the whole illness much lower. The pulse is rapid and feeble, and the patient soon becomes pallid, while the bodily strength is in many cases quickly prostrated. The appetite is lost, and feeding becomes difficult and painful from the condition of the throat. In a large proportion of cases, variously estimated at 30 to 60 per cent., the urine is albuminous, and this occurs, not after the illness, as in scarlatina, but during the height of the throat symptoms. In the majority of cases, the specific inflammation is limited to the tonsils, soft palate, and uvula, but it may spread to adjacent mucous membranes—those of the nose and the conjunctiva, the Eustachian tube, and the larynx and respiratory passages. If the *nose* be affected, there is more or less obstruction to nasal respiration, the mucous membrane is swollen, and a mucopurulent or thin brownish mucoid secretion runs from the nostrils, reddening or excoriating the alæ and adjacent upper lip. It may be streaked with blood, or decided epistaxis may occur.

Diphtheria of the larynx presents the symptoms of laryngitis, and the obstruction, due to the swollen mucous membrane, is increased by the presence of the diphtherial false membrane. The first warning is often given by the occurrence of a noisy, brassy, or *croupy* cough, soon followed by the noisy or stridulous breathing during inspiration and expiration, which indicates that the glottis is narrowed. As obstruction increases, the supra-clavicular, supra-

sternal, and intercostal spaces are sucked in with each inspiration; and in infants and young children, with soft yielding bones, the lower end of the sternum, or the three or four lower ribs, are drawn in, showing the extent to which the air is prevented access through the glottis to the lungs.

Slight degrees of obstruction may persist some days without much change, but more often the case gets progressively or rapidly worse. The face, at first flushed, with bright eyes, gets pallid, and finally livid or cyanosed. The child is restless, putting its hand to its mouth or throat, as if to remove the impediment. The cough becomes husky rather than clanging, and from time to time there may be spasmodic closure of the glottis, in which violent inspiratory efforts are made, and the cyanosis becomes extreme. In other cases the child becomes gradually drowsy and cyanotic, the skin becomes cool, drops of perspiration stand upon the forehead, and death ensues without any struggling on the part of the child.

As a rule, in these cases the diphtherial process is not confined to the larynx; it spreads to the trachea and the bronchi, forming a continuous membrane in the former, which, in the middle-sized and smaller bronchi, is gradually changed into a simply purulent secretion. These morbid products naturally increase the difficulty of breathing, though it is not always easy to recognise their presence by physical signs. Generally a loud and stridulous noise is heard in the chest, caused by the obstruction at the glottis. It may be mixed here and there with mucous râles, and there may be patches of tubular breathing, due to the broncho-pneumonia which is so frequent a sequel of the spread of diphtheria into the lungs.

If diphtheria begins in the larynx, it produces the above described symptoms of membranous laryngitis. Such cases were called *croup* before the term diphtheria was in use, but it is now known that they are in nearly all cases of diphtherial origin (*see* Laryngitis).

Diphtheria sometimes spreads beyond the respiratory mucous membranes, when these are already affected; or from other sources of contagion membranes may form on the conjunctiva, on the mucous membrane of the female genitals, or on open wounds, such as the tracheotomy wound so often required.

In the simple pharyngeal cases death takes place by asthenia and cardiac failure, sometimes with extraordinary suddenness. Occasionally, dilatation of the heart can be recognised by physical signs, and the pulse becomes quick, feeble, and irregular. It may thus happen on the second, third, or fourth day, or later. Sloughing of the tonsil or pneumonia may precede death, and enlargement of the liver has been noted in some cases.

In the laryngeal cases, death takes place from increasing asphyxia, or from an attack of spasm of the glottis; but if laryngeal obstruction has been obviated by intubation or tracheotomy, blocking of the smaller tubes, or broncho-pneumonia, may bring about a fatal result, or the patient may die from asthenia, as in the pharyngeal cases.

Complications and Sequelæ.—Complications are chiefly the

extension of the disease to different parts, which have been described. Pleurisy may accompany the pneumonia. Albuminuria is rarely more than a symptom, but occasionally a definite nephritis may persist or occur as a sequela. The lymphatic glands may suppurate or slough.

The most important sequel of diphtheria is the affection of the peripheral nerves, which results in *diphtherial paralysis*. This shows itself first in the soft palate. Some days, or a week, or several weeks, after apparent recovery, the child is noticed to speak with a nasal, twanging voice, and when it swallows liquids a small quantity is regurgitated through the nose. These defects are due to paralysis of the soft palate, which fails to shut off the mouth from the nose, as it should during speaking and swallowing. Shortly after this the child is noticed to be weak in the legs, and unable to walk any distance, or the knees give way on standing for a short time. The knee-jerk is lost quite early. In older children and in adults failure of accommodation of the eye for near objects is often noticed, due to paralysis of the ciliary muscle; and the extrinsic muscles of the eye may be also affected, producing strabismus or squint. In many cases the paralysis does not proceed beyond this stage, but in a few weeks the muscles recover their power completely. In others, the muscular system throughout the body may be affected. The patient lies motionless in bed, respiration is rendered difficult from paralysis of the intercostal muscles or diaphragm, and food given by the mouth is rejected, from inability to swallow it. The paralysis of the diaphragm is often followed by collapse of the lower lobes of the lungs (*see* Lesions of the Phrenic Nerve). The laryngeal muscles are also sometimes affected—one, or many, or all of them. Thus there may be paralysis of one cord, or paralysis of the abductors, or paralysis of all the muscles, with cadaveric position of the cords. The voice in the last case will be lost completely, and variously modified in other cases (*see* Paralysis of the Larynx).

Sensory symptoms may occur; but in children they are frequently not detected. They consist in a feeling of numbness, or formication, or distinct anæsthesia, especially in the extremities. Ataxy has been observed with very little actual paralysis; and rarely transient muscular spasms. Sometimes the muscles or the nerve trunks are tender on pressure. In severe cases electrical reactions are diminished, and some muscular atrophy ensues. Recovery generally takes place within three or four months, and the paralysis rarely, if ever, becomes chronic. Death, however, results sometimes from paralysis of the diaphragm, with gradually increasing accumulation of secretion in the bronchial tubes; and sometimes from cardiac paralysis, causing a feeble, irregular or intermittent, generally quick, but sometimes slow, pulse, with vomiting and cyanosis.

Pathology.—The inflammatory change which is characteristic of diphtheria is the formation of a membrane, which is separable with more or less ease from the affected surface. As already stated, this may be preceded by a catarrhal stage, with the secretion of mucus; and it may go on to gangrene. The membrane is the

combined result of necrosis of the superficial tissues, and the exudation of fibrin and leucocytes. In the trachea the epithelium is shed early, and the membrane consists chiefly of fibrin and leucocytes, loosely attached to the surface. In the fauces, on the other hand, the stratified epithelium is infiltrated with fibrin as well as the sub-epithelial connective tissue, and necrosis takes place causing the formation of a grayish-white or white layer firmly adherent to the deeper tissues. In the smaller bronchi the exudation is purulent; the lungs often present lobular pneumonia, with occasional hæmorrhages.

The changes in the various organs are attributable to the influences of the toxins circulating through the body. In cases where the failure of the heart has been a cause of death, its substance may be pale, soft and friable; the muscular fibres are fatty, and blood is extravasated. The kidneys present changes similar to those following scarlatina—that is, they are in a stage of moderate acute nephritis, the tubes being distended with swollen, opaque, and granular renal cells.

After diphtherial paralysis the peripheral nerve-trunks have been found extensively degenerated by Mendel and others; and Sidney Martin has shown in animals that the same degeneration of the nerve-trunks can be obtained by injecting the chemical products of diphtherial lesions into the veins.

Streptococci and staphylococci are often present in the superficial layers of the diphtherial membrane, and may sometimes lead to secondary suppurative lesions.

Diagnosis.—In early stages of a disease suspected of being diphtheria, the diagnosis can only be positively established by the bacteriological cultivation of the Klebs-Loeffler bacillus from the secretions of the affected part. This is generally done by means of a swab of cotton wool on the end of a piece of wire: the swab is smeared over the fauces or tonsil, inserted in a sterilised glass or metal tube, and sent to the bacteriological laboratory. If this is not possible, attention should be paid to the following points. The presence of a bright white patch with an inflamed areola, upon the uvula or soft palate, is generally distinctive of diphtheria; but some difficulty may occur when the tonsil alone is affected.

In *follicular tonsillitis* small yellow plugs are often seen, but there may be white plugs of secretion exactly like the diphtherial deposit. In this case, several small plugs at the same time, as contrasted with one large patch, are in favour of the follicular form. The spread of the membrane to the soft palate, albuminuria, or the co-existence of inflammation of the larynx confirms the diagnosis of diphtheria.

Vincent's angina, which is described under Diseases of the Throat, resembles diphtheria, but can be distinguished by the bacillus which causes it.

In *scarlatina* the tonsils are swollen and are covered with viscid, mucoid, and often yellow secretion; and the occurrence of a definite white patch would be generally regarded as proof of a

complicating diphtheria. In the earliest stages of throat inflammation it may be impossible to say, unless by cultivations, until the rash of the one disease or the membrane of the other is seen. As to the diagnosis between diphtheria and *croup*, it must be remembered that an apparently spontaneous, or catarrhal, *primary* laryngitis occurring in a child, even if there are no patches on the fauces, is mostly caused by diphtheria; it is certainly diphtherial if there are patches in the throat, if there is albuminuria, if the laryngitis is the result of contagion or transmits disease to another, and if it is followed (but this rarely happens) by paralytic symptoms.

Diphtherial paralysis is distinguished by the nasal voice, the return of liquids through the nose, the loss of visual accommodation for near objects, the weakness of the lower extremities, and the absence of knee-jerk. In more advanced cases dysphagia with rejection of food may be mistaken for vomiting.

Prognosis.—The mortality from diphtheria has been considerably reduced since the introduction of the treatment by antitoxic serum in 1893. In the hospitals of the Metropolitan Asylums Board during 1891, 1892, 1893, the mortality was 30 per cent.; in 1909 it was 9·8 per cent. The chance of recovery is diminished by every day, or half-day, that the treatment is delayed. Extensive formation of membrane, spread of the disease to the nose, rapid failure of strength, and feeble pulse are of unfavourable prognosis, but their occurrence is often prevented by early treatment. Diphtheria of the larynx and trachea is more fatal, because, though laryngeal obstruction may be relieved by tracheotomy, death may occur from purulent bronchitis or broncho-pneumonia caused by extension to the lungs. In these cases also the mortality has been much diminished by antitoxin. Diphtherial neuritis generally recovers, but is occasionally fatal through failure of the diaphragm.

Treatment.—Immediately upon the diagnosis of diphtheria being known, and even before, if there is a high probability of the suspicion being confirmed by the bacteriological test, *diphtheria antitoxin* or *antitoxic serum* should be injected.

The methods of preparing the serum vary somewhat in detail, but the principle is the same. An animal, for instance, the horse, is gradually rendered immune by successive injections of increasing quantities of the culture fluid of the diphtheria-bacillus, deprived of the organism itself. When the animal is at length completely insusceptible to the diphtheria poison, its blood-serum is found to have the power of neutralising the influence of diphtheria cultures inoculated into animals; and hence it appears that this serum contains a substance (*antitoxin*) which antagonises the toxin of the diphtheria-bacillus. The serum is standardised by experiment upon animals. The unit adopted by Ehrlich is the amount which, when mixed with a hundred times the fatal dose of toxin, protects a guinea-pig of 250 grammes weight from death within four days. The initial dose required is from 4000 to 12,000 units, according to the severity of the disease; and 4000 units may be contained

within 20 c.c. of the serum. The dose may be repeated at intervals of twelve or twenty-four hours during the next two days, and the amount must be estimated by the intensity of the disease, and not by the age of the patient. The injection should be made under the skin of the flank, with antiseptic precautions. An effect is very often observed in a few hours, either in the fall of the temperature or at least in the arrest of the progress of the symptoms. An urticarious or morbilliform rash sometimes follows the injections.

Treatment in general must be supporting and stimulating. The patient should be confined to bed, and good nourishing liquid food given in small quantities frequently. The fever is not often so high as to require special attention; tonics, such as ferric chloride and quinine, or tincture of cinchona and ammonium carbonate, constitute the chief part of the internal medication; but if the heart dilates, and the pulse becomes feeble, a few drops of tincture of digitalis or liquor strychninæ are desirable. With these, wine and brandy will be early required, and in severe cases they must be given freely.

Local remedies are applied partly as palliatives, partly as antiseptics or parasiticides. They are applied either to the membrane itself or to the raw surface after the membrane has been removed by forceps. Among these, nitrate of silver and hydrochloric acid, carbolic acid, and tincture of iodine may be mentioned; and pepsin, papain, and alkaline solutions have been used as solvents to the membrane itself. But it is undesirable to remove the membrane forcibly, or to apply powerful caustics. The more common applications are disinfectants and astringents, which, if they have no specific influence on the lesion, may at least check the growth of micro-organisms on the surface, moderate the inflammation, and prevent putrefaction. Lotions of permanganate of potassium (2 gr. to ℥j), chlorine water, the liquor sodæ chlorinatæ of the U.S. Pharmacopœia, formalin (1 in 200), chinosol (1 in 600), the tincture of ferric chloride (℥ss to ℥j), carbolic acid (2 gr. to ℥j), borax or boric acid (saturated solutions), may be applied every four hours with a brush, or, in somewhat stronger solution, may be used as a spray. A useful solution from the spray consists of carbolic acid, 120 grains; iodine liniment, 2 drachms; rectified spirit, 1 drachm; water to 12 ounces.

For the removal of the offensive and irritating secretions, when the nasal mucous membrane is involved, the nostrils should be syringed with dilute disinfectant solutions, such as potassium permanganate and carbolic acid; or these may be administered by the nasal douche.

When the larynx is attacked the patient should be subjected to an atmosphere saturated with moisture. In a small room it will be sufficient to use a bronchitis kettle, the steam from which may fill the room. Much relief is also sometimes given by a hot bath. If improvement is not apparent in a few hours intubation

or tracheotomy should be performed, and this must be done at once if there is sucking-in of the chest, if the patient is drowsy, or becoming cyanosed, or if the forehead is cold and clammy. The probability of success is greater the earlier a tube is introduced into the larynx or trachea; and if a case is seen from the first the above indications of carbonisation should be anticipated, and the operation should be done while the child is strong and of good colour. Nevertheless, success sometimes follows under most adverse circumstances; and even the existence of pneumonia should not deter one even from opening the trachea. Generally in diphtheria tracheotomy is to be preferred to intubation. The latter is bloodless, and if it fails can be succeeded by tracheotomy; but it requires special skill in its performance, and the risk is run of pushing membrane down into the trachea. Nearly always some improvement follows an operation; the child breathes freely and deeply and sleeps tranquilly; but the danger still remains of pulmonary complications, which may be fatal in a few days after the operation. As already stated this danger is much lessened by the use of antitoxin in these cases. Internally, expectorants, such as ammonia or ipecacuanha in small doses may be tried; and in cases with much discharge of membrane its expectoration has been facilitated by frequently spraying down the tube with solutions of sodium bicarbonate (20 grains to 1 ounce). Probably the moisture has been as efficacious as the solvent. The tracheotomy tube may often be removed in from one to four days.

Diphtherial paralysis generally passes off in from two to four months. Rest, tonics, and electricity are desirable. In the more severe cases, where swallowing becomes difficult, feeding by the nasal tube, or by nutrient enemata or suppositories, may be necessary. Diaphragmatic paralysis should be treated by galvanism, one pole being applied over the phrenic in the neck, the other to the epigastrium. Oxygen gas may be inhaled at intervals, and ammonium carbonate should be given in frequent doses, *e.g.*, for a child 1 to 3 grains every two hours, to relieve the accumulation of mucus in the chest.

Prevention.—The same problem as engages us in the case of scarlatina has here to be solved: namely, that the virus may persist in the body long after the patient is himself quite well, and hence the risk of contagion remains. It is usual to keep a diphtheria patient from contact with others until bacilli can be no longer cultivated from the throat or nasal secretions. Sometimes a period of several weeks elapses before the patient is free. As in typhoid fever, also, there occur *diphtheria-carriers*, in whose throats the bacilli are latent, harmless to their hosts, but causing infection in others with whom they come in contact. It is here important to remember that bacilli are sometimes cultivated from the throat, both together with the Klebs-Loeffler bacillus and apart from it, which resemble it closely, but, unlike the B. diphtheriæ, are not virulent to guinea-pigs. The most important of these is *Hofmann's*

bacillus, which is often found in the throat. It is about 2μ in length, and generally arranged in pairs; and it produces alkali, not acid, in milk and glucose media. Though mostly regarded as distinct, the possibility of its conversion into a virulent B. diphtheriæ has been entertained.

CHOLERA

(*Asiatic Cholera*)

The name Cholera is now almost exclusively given to an acute specific disease, of which the principal features are the profuse discharge of watery evacuations from the bowels, vomiting, collapse, cramps in the calves and feet, and suppression of urine. It is constantly present in India, where even in the present day hundreds of thousands die of it (400,000 in 1907, 590,000 in 1908). It has spread from time to time during the last hundred years to Europe and other parts, but though the extraordinary fatality of these epidemics has made of each occurrence in different countries a historical event, the disease has never obtained a permanent hold in this continent. In 1830 it was epidemic in Europe, and in 1832 invaded the British Isles. Thence it reached the New World, and was again brought back to Europe. England again experienced severe epidemics in 1849 and 1854, and a milder and more restricted outbreak in 1866.

Koch described in 1883 the *comma bacillus* or *spirillum* of cholera. It is found in the rice-water evacuations, in the contents of the intestine after death, and in the mucous membrane of the intestine, just beneath the epithelium; but it has not been found in the blood. It is a little shorter than the tubercle bacillus, slightly curved, somewhat thicker in the middle than at the ends, and flagellated at one or both ends. It can be cultivated in the usual media, and though spirilla which closely resemble it are found in other conditions, the evidence in favour of its specific relation to cholera is very strong. Among other facts are these: that the serum of convalescents from cholera agglutinates the comma bacillus; that it is protective against inoculation of animals with human spirilla; and that the human subject can be immunised by cholera antitoxins.

Ætiology.—Cholera closely resembles enteric fever in the way in which it is conveyed—that is, there is rarely, if ever, direct contagion from man to man, as in scarlatina and small-pox; but the *materies morbi* is present in the evacuations, and it is by means of these contaminating water used for drinking, cooking, or washing, that its entrance into other individuals is effected. It is sufficient here to refer to the historical Broad Street Pump, which in 1854 was the cause of a severe epidemic. The epidemic was stayed when the pump was padlocked; and it was subsequently shown that the discharges from a patient, who had contracted cholera elsewhere, had found an entrance into the soil from which the pump-water was drawn. In 1866 the epidemic in London mainly

affected the eastern portion, supplied with water by the East London Waterworks. A reservoir belonging to the water company was found to have been contaminated with sewage materials, filtering through the soil from a leaky sewer pipe; and this sewage was infected with cholera by the dejecta of a patient previous to the general outbreak. The spread of the disease in India is often determined more or less by the direction of the rivers. As in typhoid fever, the influence of flies has to be considered: and the occurrence of outbreaks due to *cholera-carriers* (*see* pp. 42, 120, 127) is now clearly recognised.

Although in India the Sepoys living on the lower levels are less susceptible to it than the hill tribes, in epidemics affecting Europeans and others it makes but little distinction of sex, age, or condition. It is rare, but occasionally occurs, at high altitudes. Summer and autumn are the most favourable seasons; and it is promoted by alternating dry and wet weather, and checked by protracted drought or excessive rains. Its spread is stopped by cold, but it will survive the winter and break out again in the spring or summer. Individual predisposition is shown especially in poverty, malnutrition, and chronic alcoholism; and the onset seems sometimes to have been determined by excesses in diet.

As a rule, one attack of cholera is protective against a second.

Course and Symptoms.—The *incubation* is mostly from two to three days, but may be a week or two; exceptionally it is less than two days. There is sometimes a *prodromal* or *premonitory* stage of diarrhœa, or in the absence of diarrhœa, the patient is depressed and uncomfortable, and complains of headache, vertigo, noises in the head, or oppression at the epigastrium; and this stage lasts from one to two or three days. Then the patient is seized with violent diarrhœa, and the discharges soon lose all biliary colouring-matter, and look like whey, or water in which rice has been boiled (*rice-water stools*). These are neutral or slightly alkaline, of sp. gr. 1006 to 1013, containing sodium chloride, albumin and mucin. If allowed to stand they deposit a finely granular whitish-gray sediment, consisting of epithelium, leucocytes, shreds of tissue, crystals of ammonio-magnesian phosphate, bacteria, comma-bacilli, threads of algæ, and blood-corpuscles. Sometimes the stools have a pink tinge from admixture of blood. The purging is accompanied by borborygmi and gurgling, but by little pain or griping. After one or more hours of purging, vomiting sets in; at first food is rejected, then a whey-like fluid, like the intestinal discharges. The vomiting is easy, often a mere regurgitation. The patient suffers from anorexia and thirst, the tongue is white and may become dry, and the epigastrium is sensitive to pressure. About the same time, in most cases, there are severe and extremely painful cramps in the calves of the legs, in the feet, and less often in the hands and trunk. Soon the patient sinks into *collapse*—the *algide* stage. The surface of the body becomes cold and livid, the hands, feet, face, and nose are pinched and blue, the eyes are sunken, and the breath

is cold; the axillary temperature falls 4° or 5° below the normal, while in the mouth it may be even lower still. On the other hand, in the rectum and vagina it has been found to be 102° or 104° during cholera collapse. In severe and fatal cases the eyes become dry and the cornea cloudy. The pulse is small, thready, almost imperceptible, numbering from 90 to 100. Respirations are short and quick, from 35 to 40 in the minute. There is great muscular prostration, but the patient is restless, throwing his limbs about; the voice is hoarse, or sinks to a whisper (*vox cholerica*), or only the lips are moved in the attempt to speak. Purging often ceases during collapse, but vomiting continues. The urine becomes scanty, and is often entirely suppressed—a condition which may begin quite early, and last thirty-six or forty-eight hours; it is probably only a result of the failing circulation. The patient generally retains complete consciousness, though lying apathetic and indifferent, except when aroused by the pain of cramp. This stage begins six or seven hours after the first symptoms, and lasts twelve or twenty-four hours, when the patient may die without rallying.

In cases which survive the collapse there is a gradual rise of temperature, the skin begins to regain its natural colour, and loses its shrunken appearance, the cramp and restlessness cease, the pulse improves, and may become slower than in health, urine is again secreted, but contains less urea than normal, and frequently albumin and casts. The face becomes congested with patches of dusky redness, and the conjunctivæ are injected. This is described as the stage of *reaction*, and goes on to recovery.

Varieties.—The variations from this more usual course are many. Milder forms occur in which the disease does not pass beyond the first stage of diarrhœa. Sometimes diarrhœa does not even confine the patient to bed, but bacilli are nevertheless found in the motions. A more severe form, *choleraic diarrhœa*, begins suddenly, after exposure to cold or some error in diet, with profuse but painless diarrhœa, the motions being abundant, fluid, of yellow, or yellowish-brown colour, containing epithelium, crystals of ammonio-magnesian phosphate and bile constituents. There are from two to six or eight motions in the day, and they are attended with borborygmi, and sometimes with cramps of the calves. The diarrhœa lasts from a few days to one or two weeks.

Cholerine, a still nearer approach to the severer attacks, also occurs suddenly and unexpectedly, vomiting accompanies the purging, the motions often become colourless, and there may be cramps, some cooling of the extremities, scanty urine, and albuminuria. Recovery is slow.

Sometimes the stage of *collapse* may set in at once, and the patient may sink before any purging has taken place at all (*cholera sicca*). And in other cases beginning as usual with the diarrhœal stage, the collapse may be very short, the patient dying suddenly from respiratory or cardiac failure; or in others, again, the algide condition is prolonged to thirty-six or forty-eight hours.

In other cases the *reaction* is imperfect: either a relapse occurs, with purging, vomiting, collapse, and even a fatal result; or diarrhœa continues, or vomiting, or sleeplessness. Even in the stage of reaction the temperature is not generally above the normal, but there is sometimes slight pyrexia, and with this a *rash*, erythematous, roseolous, or urticarious, which commonly begins on the hands, backs of the forearms and feet, and spreads to the trunk (*roseola cholerica*). It appears at the end of the first week, or in the second week, and lasts from two to four days. A more serious development of this stage is the so-called *cholera-typhoid*, which occurs about the end of the first week. There is great prostration, with headache, flushed face, coated tongue, loss of appetite, nausea, or vomiting; the bowels may be loose or confined. Vertigo is frequently complained of, and the patient becomes drowsy or comatose. The temperature rises to 100° or 101°, the pulse is weak and small, and there is mild delirium at night. The urine is albuminous. Cases, moreover, in which fatal coma occurs, preceded by stupor, restlessness, muttering delirium, and muscular spasms, are regarded as uræmic. The condition lasts from two to nine days, ending in recovery, or in death by coma.

Complications.—The following have been observed during the typhoid stage, or during convalescence: bronchitis, œdema of the lungs, pneumonia, or pleurisy; diphtherial inflammation of the pharynx and larynx, bladder, and female genitals; meningitis; arthritis; parotitis; gangrene of the scrotum and penis, or of the nose; conjunctivitis, and opacity and ulceration of the cornea from exposure during the stage of collapse; and bed-sores.

Pathology.—Decomposition proceeds slowly in those dead of cholera, and *rigor mortis* persists a long time. The right side of the heart is often distended with blood, the lungs are engorged, and the mucous membrane of the trachea and large bronchi is congested. There is often purulent mucus in the minute bronchi, and in cases dying in late stages there may be hæmorrhagic infarcts. The *intestine* contains in earlier stages rice-water fluid, in later stages liquid tinged with green. The mucous membrane is congested, and frequently sodden and pulpy: and Peyer's patches and the solitary follicles are swollen and prominent. The lower end of the ileum is the part most affected. The *spleen* is small. The *kidneys* are large, and show proliferation of the epithelium and cloudy swelling. Later on, casts form in the tubes, the organs become paler, and fatty degeneration takes place.

Diagnosis.—Cholera, in its milder forms, may be exactly simulated by the severer kinds of diarrhœa from gastro-intestinal irritation and food-poisoning, in which there may be pronounced collapse, prostration, scanty urine, and loss of colour in the motions. These cases are generally single or in family groups (*see* Enteritis). Arsenical poisoning, producing similar symptoms, has been also mistaken for cholera.

The final appeal is to bacteriological tests and microscopic

examination. For the latter purpose portions of intestinal mucus may be stained on a warm stage with a weak, fresh, watery solution of methyl-violet; or a dried film may be stained with fuchsin.

Prognosis.—The mortality in epidemic times varies from 40 to 60 per cent. It is more fatal to the very young and to the aged; to those who are in ill-health, are debilitated by insufficient nutriment or bad hygienic conditions, or are the subjects of chronic alcoholism. The unfavourable symptoms are profuse and violent discharges, rapid prostration, with much cyanosis, shrivelled and cold skin, profuse cold perspiration, and absence of pulse at the wrist.

Treatment.—It has been the general practice to treat with opiates and astringents the diarrhœa which is prevalent during epidemics of cholera. These should be used in the premonitory period, but they do no good in the pronounced stages of the disease itself; and excessive medication in any form may do harm, since whatever is introduced into the stomach will either be vomited again or will lie unabsorbed from the inactive condition of the circulation. Even stimulants, such as brandy, ammonia, or ether, must be given in small quantities and with caution. Some relief may be obtained from sucking ice or taking small quantities of ice-cold water or of effervescing draughts. Severe cramps may be treated by small doses of morphia subcutaneously or by gentle friction with chloroform or stimulating liniments. Cold compresses will relieve the præcordial distress. The condition of collapse must be met by supplying heat by means of warm blankets and hot bottles to the feet or the sides of the chest or thighs or by hot baths. The intravenous injection of normal saline solution (sodium chloride ·7 per cent., or a drachm to a pint) should be tried in cases of profound collapse.

In the stage of reaction the patient should be kept cool; diarrhœa, if it continues, may be checked by opiates or astringents; the vomiting, by effervescing draughts or by opium. Light, nutritious food should be given frequently in small quantities. With much vomiting it may have to be given *per rectum*. Continued suppression of urine will require small doses of salines, and counter-irritation or cupping-glasses to the loins.

Prevention.—Since cholera epidemics are spread mainly by the discharges from the stomach and intestine, infecting the air, food, or water-supply, the greatest possible care must be taken to prevent any such contamination when a case is first recognised. The more perfect the sanitary arrangements with reference especially to water-supply, drainage, and the removal of sewage, the less likely is this to take place. In all these respects cholera and typhoid fever (*see* p. 42) are precisely the same. Choleraic discharges cast into sewers without previous disinfection render the contents infectious, and may poison drinking water when the drains, sewers, or wells are faulty. The same discharges soiling bed-linen, clothes, towels, and similar things may directly or by infecting washing-water be

the means of conveying infection to remote parts. The above indicates the direction in which one should act: disinfection of the discharges, and of everything soiled with them, and constant watchfulness to avoid contamination of water, as well as air- and food-supply. Cleanliness in every way helps in this respect. Quarantine, as understood on the Continent, has long been distrusted by English authorities, and the regulations issued with regard to vessels entering British ports only provide for thorough inspection by the medical officer of health, who has to see that such means are taken by disinfection of the vessel, and by the removal and detention, under proper treatment, of any that may be suffering from cholera, as will prevent its spread to healthy individuals.

Prevention has been attempted in another form of late years—namely, by inoculation of healthy individuals with cultivations of the cholera-bacillus. M. Haffkine has elaborated a method, which has been used extensively in India, with promising results. He injects first an attenuated culture of comma-bacilli, obtained by growing them on artificial media, continually aerated; and then a stronger ("exalted") culture prepared by inoculating the peritoneal cavities of a series of guinea-pigs. As each animal dies, the fluid from its peritoneal cavity is used to inoculate the peritoneum of another, and so on until twenty or thirty animals have been employed, when the maximum virulence, estimated by the shortness of the period between inoculation and death, has been reached. One c.c. of the weak "vaccine" is first injected into one flank, and after four or five days one c.c. of the "exalted" vaccine is injected into the opposite flank. Headache, fever, and lassitude of three or four days' duration follow each injection.

PLAGUE

In the Middle Ages this term was used to designate any severe or fatal epidemic, but as now understood its meaning is restricted to one particular disease, the Bubonic, Oriental, or Levantine plague. This is an acute febrile disease, usually attended by swelling of the lymphatic glands in the groin or other part of the body, but sometimes fatal without such lesions. Its history can be traced back to the second century of the Christian era, but the first great epidemic in Europe, the plague of Justinian, occurred in the sixth century. Epidemics were frequent in the Middle Ages; but since the year 1665, when London was devastated by the plague, these epidemics have gradually become less frequent in Europe, and in the first third of the nineteenth century were confined on this continent to the most easterly portions of the Turkish Empire. Recently the plague has again become of importance to European peoples, from its occurrence in Hong Kong and South Eastern China in 1894, and in Bombay and other parts of India in 1896; from which sources the disease has occasionally made its appearance, and for longer or shorter time in different ports of

Europe, South Africa, and Central and South America. In India for the thirteen years 1896–1908 it caused more than six million deaths; and in the first ten months of 1910 the number was over 380,000. Some cases occurred in England in the same year.

The plague is a specific disease, due to a bacillus (*B. pestis*) discovered by Kitasato, which may be found during life in the blood, in the inflamed glands, in the fæces and urine, and in the sputum of certain cases; and after death in almost every organ of the body. The bacillus is a short rod, with rounded ends, measuring from 1μ to 1·5μ in length, flagellated, and staining more deeply at the ends than in the centre.

Ætiology.—Though direct contagion through the respiratory organs occurs in the pulmonary form, it is probable that the bubonic form is rarely contracted in this way, but much more often by infection through the skin. This is suggested by the frequency with which the superficial groups of glands are the first to be involved. There is now very strong evidence in favour of the view that the disease is conveyed to man from infected rats by the agency of fleas. It has long been observed that rats have plague, and often die in great numbers in any community before human beings are attacked. And now plague bacilli have been found abundantly in fleas taken from plague-infected rats; fleas certainly convey plague from rat to rat; rat-fleas experimentally have been known to bite a man's hand and to live for days upon his blood (Advisory Committee's Second Report); and patients with plague have been found to be flea-bitten. It is possible, however, that the mucous membranes of the nose and mouth, and even genital organs, may also be channels of entrance: but rarely the stomach or intestines. Infection of dwelling-houses, and clothes by sputum, fæces, or urine has also been regarded as contributory.

The influence of season and climate is variable: very great heat seems to have checked the disease with more certainty than cold.

The disease attacks people of all ages up to fifty, after which year it is much less common. One attack confers a relative immunity from others.

Symptoms.—Several varieties of plague are now recognised. The more common form is the *bubonic*, which is characterised by glandular enlargements. The *incubation* is from two to five days: and the disease begins with lassitude, weakness, headache, vertigo, and shivering, soon followed by febrile reaction. Sometimes in this stage of invasion the patient is in a peculiar absent condition, with staggering gait and tremulous speech: or he is seized with indefinable fear and restlessness; or there may be nausea, vomiting, or diarrhœa. The fever is generally high, the temperature from 102° to 104°, or in the worst cases over 107°, the pulse from 100 to 130. The tongue, at first moist and white, becomes dry and brown, and a typhoid condition may supervene with delirium or coma, sordes on the lips and teeth, failing pulse and cold extremities. The urine is scanty, acid, of high colour, and it usually contains

albumin: and suppression occurs in some fatal cases. After one, two or three days' fever the local signs show themselves in the formation of glandular swellings in the groins, axillæ, or neck. Mostly only one group is swelled, and generally it is the inguinal glands that are affected. The swelling may be as large as a hen's egg, or larger, is attended with severe pains, and if the patient survives may suppurate about the seventh day. About this time also boils or carbuncles may appear, but they are not very frequent; they occur on the lower extremities, the buttocks, or the back of the neck. In the severest cases petechiæ, or larger subcutaneous hæmorrhages, appear shortly before death, either distributed generally over the body, or more marked in the neighbourhood of the enlarged glands; and there may be bleeding from the nose, lungs, stomach or bowels. Death takes place mostly before the sixth day. In cases that recover, convalescence begins from the sixth to the tenth day, but may be much protracted by suppuration of the glands.

In *Septicæmic plague* the patient is struck down with great rapidity, and may be dead in twenty-four hours. The lymphatic glands may be somewhat swollen, but no large buboes form. The pulse rapidly fails, hyperpyrexia may occur, and delirium and coma end the scene. This no doubt includes cases formerly described as *fulminant* (*Pestis siderans*).

Pneumonic plague.—This is an important variety, which was first recognised in Bombay in 1896. It begins like the bubonic form, but within a day or two respiratory symptoms become urgent: there are very rapid, shallow breathing, cough, expectoration of much sputum tinged or streaked with blood, sleeplessness, restlessness, early delirium, and death within three days. The physical signs of consolidation are not prominent, but râles and rhonchi are present. After death patches of pneumonia are found scattered through the lung, and buboes are usually absent. The bacillus is found in the pneumonic patches and in the sputum.

Pestis minor and *pestis ambulans* are varieties in which the fever is slight and the symptoms are mild, so that sometimes the patient may walk about during a great part of the illness.

Morbid Anatomy.—The enlarged lymphatic glands are found to be inflamed, red or violet in colour, soft or pulpy in consistence, and surrounded by connective tissue infiltrated with serum or blood. The internal glands in the same neighbourhood are involved; thus, the pelvic glands with inguinal buboes or the mediastinal glands with buboes of the neck. The liver is congested, the spleen is large and dark, and there are ecchymoses of the mucous and serous membranes, and swelling of the kidneys with hæmorrhages under the capsule and sometimes into the surrounding cellular tissue. The lungs show patches of consolidation in the pneumonic form, but in the other varieties only œdema and minute hæmorrhages; there is often some pleural effusion.

Diagnosis.—This is especially difficult with the first cases

imported into a new district, and such early cases have been mistaken for yellow fever, typhoid, typhus, or malaria; and confusion with diphtheria, parotitis, and gonorrhœal bubo has also occurred. The distinctive early symptoms are the expression of the face, the hesitating speech, and staggering gait, and later on the numerous buboes. But the ultimate diagnosis must depend on the detection of the bacillus, which may be found in the blood, in the juice or pus from buboes, or in the sputum. The contents of buboes may be removed with a sterilised glass pipette, placed on a slide or cover-glass, gently heated to dry and fix, coloured with carbol-fuchsin or gentian violet, and examined with a $\frac{1}{12}$-in. oil-immersion lens.

Prognosis.—The mortality has usually been very great, reaching 40, 50, or even 80 per cent.; but it has varied in different towns, and is less among white men with good sanitary arrangements than under opposite conditions.

Treatment.—This is for the most part symptomatic, and pain, collapse, or hyperpyrexia must be dealt with, as in other acute specific diseases, by opium, stimulants, local application of cold, &c. Cantlie recommends the early use of calomel and purgative salines. Several kinds of antiplague serum, obtained in the same way as the antitoxic serum of diphtheria (*see* p. 125), have been tried in plague. One of the best is that of Yersin and Roux: in two series of cases treated with it, and without it, the apparent reduction in mortality was 10·5 per cent.

Prevention.—This comprises notification of the disease; the isolation of any patient suffering from the disease; the disinfection of dwelling-houses, clothes and bedding, preferably by solutions of perchloride of mercury; the disinfection of persons taken from infected houses; the destruction as far as possible of rats. Haffkine has employed as a prophylactic the injection of a broth culture of plague bacilli, of which the bacilli have been killed by heat; and the dose has been regulated with the view of obtaining an average temperature of 101° in the inoculated person. A considerable degree of success has attended its use. Calmette prefers a preparation of bacilli washed free of toxins and dried.

SYPHILIS

Syphilis, or The Pox, is a specific infectious disease, conveyed by inoculation, and producing successively a lesion at the seat of inoculation (*primary* lesion); lesions of the skin, mucous membranes, and other parts after an interval of a month or more (*secondary* lesions); and, after one or more years, deeper lesions of the skin, bones, muscles, and viscera (*tertiary* lesions). Still later results (*parasyphilitic* lesions), which differ anatomically from the earlier lesions, are now recognised in certain diseases of the nervous system and arteries, namely, locomotor ataxy, paralytic dementia, atheroma, and aneurysm.

Syphilis can be transmitted from parents to children, and then

takes on forms which differ in some particulars from the disease acquired in the usual way (*hereditary* or *congenital* syphilis).

The micro-organism of syphilis is the *Spirochæta pallida* (or *Spironema* or *Treponema pallidum*), first described in 1905 by Schaudinn and Hoffman. It is a long thin filament, of spiral or corkscrew shape, with from six to fourteen coils, and tapering at the ends to a sharp point. The length is from 4μ to 20μ, the breadth about ·25μ, and it is stained a rose-pink with Giemsa's stain. It is regarded as belonging to the class of protozoa, and is probably allied to the trypanosomes. It has been found by numerous observers in chancres, buboes, and skin papules of primary and secondary syphilis and in condylomata and other lesions of congenital syphilis. On the other hand, it is rarely found in tertiary lesions, and it is not easy to find after full mercurial treatment. In the case of superficial lesions it is frequently accompanied by an allied organism, *S. refringens*.

ACQUIRED SYPHILIS

Infection.—Syphilis is, as a rule, communicated during sexual intercourse, the delicate epithelium of the mucous membranes brought into contact allowing of the easy transmission of the virus. Cracks or abrasions of the mucous membrane do not seem to be necessary for the reception of the poison, though they must undoubtedly favour it. Syphilis may also be transmitted in other ways—for instance, in the act of kissing, by smoking pipes previously used by syphilitic persons, or by contact of syphilitic sores or secretions with the abraded finger of the medical man. Inoculation by sexual intercourse takes place commonly, in the male, on the glans penis or prepuce; in the female, on the vulva, labia, or vaginal mucous membrane. After inoculation there is usually a period of *incubation*, varying from two to nine weeks, the average being five, during which no change whatever may be observed.

Primary Lesion.—The first sign is a small red itching papule, which gradually enlarges in all directions like a flat button, and becomes very hard. The surface is dry, or scaly, or superficially ulcerated and covered with a crust of dry secretion. This condition of induration, which is most important, is reached in a week or ten days from the first appearance of the papule; and the lesion is known as the *hard, indurated*, or *Hunterian chancre*. On the mucous membrane the lesion may be scarcely so well marked; it begins as a vesicle with a red base, the vesicle breaks, and forms a shallow ulcer, the floor of which becomes indurated. In the course of time, and it may be some months, the induration gradually disappears, the ulcer heals, and a patch of pigment is left behind for a while. Though this is the typical course of the primary lesion in syphilis, the poison is often produced or is present in a lesion of a different character—the *soft chancre*. It begins as a pustule, which

breaks in two or three days, and forms a deep irregular ulcer, which has no hard base, and secretes pus freely. This pus is inoculable in the neighbourhood, and such soft sores are frequently multiple, whereas the hard chancre is generally single. The soft chancres are venereal in origin, but may be produced by other causes than syphilis; nevertheless, they sometimes contain the syphilitic poison, and all the later results of constitutional infection may then follow without the occurrence of any induration. As a further complication of this stage, it should mentioned that hard sores may, by the irritation of caustics, be made to suppurate freely, so as to simulate soft sores. During the first stage of syphilis, the poison is transmitted to the *glands* of the groin; these become indurated, and slightly enlarged, but remain freely movable upon one another, without adhesion or reddening of the skin, and do not, as a result of the hard chancre, undergo suppuration.

Secondary Lesions.—These are noticed from four to six weeks after the stage of induration of the chancre, and may continue to appear at any time up to twelve months. The most constant are certain eruptions on the skin (*syphilodermia, syphilide*), faucial inflammation, and enlargement or induration of lymphatic glands; others are febrile reaction, pains in the temples, back, or limbs, swelling of the joints, iritis, and falling of the hair.

Eruptions.—Important differences are to be noted between the early or secondary rashes, and the late or tertiary eruptions. The former are generally abundant, affecting a large part of the body, trunk, or face, and symmetrically arranged; the separate elements are more or less discrete; the lesions are superficial, do not ulcerate, and leave no scars. The latter are few, scattered, unsymmetrical, often affect the deeper layers of the skin, and form considerable ulcers, which are followed by scars; sometimes even, as in lupus, scars are formed without preceding ulceration. The elementary lesions (*see* Diseases of the Skin) of early syphilitic rashes are very variable; they may be maculæ, papules, nodules, scabs, vesicles, or pustules; and the lesions are not infrequently mixed in the same cases, constituting the feature of *polymorphism.*

The most common form of "secondaries" is the *macular* syphilide, which forms dusky pink or reddish-brown spots, rather thickly grouped, so as to form a mottled rash on the face, chest, back, and limbs. It is very variable in its duration, but may persist some weeks. It does not itch. Other forms are a *follicular* syphilide, consisting of small papular elevations, with a hair in the centre of each; a *papular* syphilide, with elevations, flat, or hemispherical, or more prominent still, so as to form nodules or tubercles, which come out in crops, irregularly over the whole body, or grouped in clusters; a *pustular* form in which successive crops of pustules occur, and are often accompanied by fever, the pustules drying up into brown or brownish-green scabs, and leaving behind slight scars; a *squamous* form, which differs from ordinary

psoriasis in the scales being less thick or shiny, in the coppery tint of the rash generally, and in the patches not being seated on the knees or elbows, or specially on the extensor surfaces; a *vesicular* and a *pigmentary* form, both of which are rare. As a special form of papule may be mentioned the mucous patches (*plaques muqueuses*), or flat warty growths which occur about the genitals, perineum, and anus, in the axillæ, groins, and under the breasts, and at the angles of the mouth—in any place, indeed, where the skin is thin and constantly moist. They are often rather extensive, with well defined edge, moist surface, and dirty-gray secretion.

Sore Throat.—Coincidentally with the rash, or even before it, the throat becomes affected; there is a diffused redness of the fauces, with enlargement and excoriation of the follicles; but the most characteristic feature is the swelling and symmetrical ulceration of the tonsils. The ulcers are often kidney-shaped, superficial, with grayish borders, painless, and not of very long duration. Sometimes, however, the tonsillar ulcers are much more persistent, extend to the soft palate and uvula, have bright red edges, and are covered with a yellowish-gray secretion, the removal of which is followed by bleeding. Other changes in the mouth in the secondary stage are white spots, like those produced by the application of nitrate of silver, mucous patches on the tongue or cheeks, bald patches on the tongue from the destruction of the papillæ, or enlargement of the tongue, which is of bright red colour, with hypertrophied papillæ, or irregular prominences, and deep sulci between them: this last condition is aggravated, or in part caused, by excessive smoking.

The *lymphatic glands* are enlarged, especially in the groins, above the inner condyles, and at the back of the neck. The *fever* of constitutional syphilis is often entirely absent, or it is represented by no more than a slight malaise or indisposition preceding or during the outbreak on the skin. In a small number of cases there is very distinct intermittent or remittent *pyrexia*, the temperature highest in the evening; and it may last for some weeks. The *hair* may come off in considerable quantities from the scalp, as well as the surface of the body and limbs; but it is not common for complete baldness to occur. The nails are sometimes affected in their nutrition also. The *periostitis* of secondary syphilis is slight; pains and tenderness are felt in the tibiæ, skull-bones, or clavicles, but they are of short duration, and nodes do not generally form as in the tertiary stage. The *joints* are not often affected; but there may be synovial effusion, which is sometimes excessive (hydrarthrosis), and is liable to vary from time to time in the same joint.

The most common affection of the eye is *iritis;* it usually affects one eye before the other; the symptoms are photophobia and pain, with ciliary congestion, irregular pupil, obscured iris, and, in severer cases, nodules of rust-coloured lymph and blocking of the pupils. Iritis occurs from three to six months after contagion:

at a later period still, but within the limits of secondary symptoms, there may be diffuse retinitis or choroiditis.

Various nervous affections, especially myelitis, are apt to occur within a few months or a year of infection, and thus fall within the category of "secondary" results. Such disorders show a considerable proportion of recoveries under antisyphilitic treatment; but, pathologically, they present either inflammatory changes not peculiar to syphilis, or at most arterial changes of a specific character.

Some other lesions occur at a time which is intermediate between the second and tertiary periods, such as scaly or peeling patches on the palms of the hands (*psoriasis palmaris*); enlargement of the testis with perhaps nodular deposit in the epididymis; choroiditis and retinitis; and transitory visceral changes, not due to gumma—for instance, enlargement or tenderness of the liver and spleen, with failure in the blood-making process, slight and temporary albuminuria, and symptoms of impending pulmonary mischief. And, indeed, no hard-and-fast line can be drawn between the end of the secondary and the beginning of the tertiary stage.

Tertiary or Late Lesions.—These are first observed from one to two years after contagion, and may recur at intervals for ten or fifteen years, or more. They are certain eruptions on the skin, periostitis and nodes on the bones, and growths in the subcutaneous tissue, muscles, meninges, liver, spleen, testis, and other viscera.

Late Syphilides.—The special features of these have been already mentioned (*see* p. 138). They are variable, and may consist of maculæ or scaly patches. But the most characteristic is a dusky red, infiltrated patch, forming a circle or broad band curved in a half-circle or horseshoe; part of the surface is covered with a brown or greenish scab, beneath which are deep ulcers with sharply-cut edges. The lesion spreads in serpiginous lines by the formation of fresh infiltrations or nodules, which in turn ulcerate, while the old ulcers heal and leave scars surrounded by deeply pigmented skin. Sometimes such nodules will subside and leave scars even without ulceration, and altogether there is a general resemblance to lupus. Ultimately large, irregular patches, of several inches in diameter, may form, and they are frequent on the knee, thigh, shoulder, forearm, face, and neck. Sometimes much deeper infiltrations of the subcutaneous tissues occur.

The growths in the viscera and other parts, which are so characteristic of the later stages of syphilis, are known as *gummata*. They consist of a substance like granulation-tissue, with a varying proportion of cells. In early stages they are gray, gelatinous and transparent; but the cells undergo fatty changes, and caseation takes place, so that the centre becomes yellow, and the circumference develops into fibrous tissue, which contracts like that of a scar. Sometimes gummata break down completely, and suppuration, with destruction of the tissue in which they are situated,

takes place; thus caries and necrosis not infrequently follow nodes on the bones. In the liver, gummata form large, more or less uniform, yellow nodules; or a yellow caseous mass lies in the centre of a fibrous cicatrix, or nothing is left but the fibrous cicatrix, with consequent depression and puckering of the organ. In the testis, gummata also occur; but this organ is often enlarged by effusion into its substance generally, and may afterwards atrophy from the formation of a dense fibrous tissue without any local nodular growth. For the clinical results of these lesions the reader is referred to the diseases of different organs. It will be sufficient here to say that gummatous periostitis, or nodes, occur especially along the anterior surface of the tibia, on the frontal and parietal bones, and on the clavicles. The patient suffers from pains, which are worse at night, and there may be found, on the affected part, flat, round prominences, from half an inch to an inch in diameter, soft, or even fluctuating, and very tender. This is not necessarily a sign of pus being present, as quite distinctly fluctuating nodes may entirely disappear under treatment. Gummata are sometimes found involving the synovial or perisynovial tissues of joints. A very definite *pyrexia*, with the temperature rising to 101° or 102° in the evening, and falling to 98° or 99° in the morning, may accompany gummata apart from suppuration. Several disorders of the nervous system are referable to syphilis; some, like hemiplegia, are due to syphilitic disease of the arteries (*obliterative endarteritis*) and consequent softening, which, occurring in the brain, gives rise to hemiplegia, or in the spinal cord to acute or chronic paraplegia, including Erb's *syphilitic spastic paraplegia;* another is probably a true infective myelitis; others, like nerve paralysis and localised convulsions, are due to gummata or meningeal thickenings on the surface of the brain and the roots of nerves. The mucous membranes are affected with deep-seated destructive ulcerations, such as we see in the mouth, destroying the uvula and soft palate, with adhesion of the remainder to the pharynx; or in the trachea, bronchus or rectum, leading to stricture or stenosis of these passages. Lastly, late syphilis is one of the causes of the lardaceous degeneration, even without the existence of any suppuration.

Parasyphilitic Lesions.—This name has been given to certain pathological conditions, with accompanying symptoms, occurring late in the history of syphilitic patients, the association of which with syphilis was first determined by statistical evidence, and not by anything characteristic of that disease in the tissue-changes themselves. These, indeed, are not gummatous, nor different from what occurs in other circumstances, but are of degenerative or chronic inflammatory nature, such as might be due to toxic influences. Moreover, these affections are much less amenable to the therapeutical actions of mercury and the iodides. But it is found that syphilis is an antecedent in from 70 to 80 per cent. of cases of locomotor ataxy and general paralysis of the insane; while some forms of ophthalmoplegia, loss of pupil light reflex, atheroma

of arteries, and aneurysms show similar relations. The correctness of this view receives support from the results of investigation of such cases by the Wassermann test (*see* p. 143).

Course and Termination.—The development of the disease varies considerably and is largely influenced by treatment. Thorough treatment in the early stages may entirely prevent the occurrence of late symptoms; and the secondary symptoms may be avoided or rendered extremely mild by diligent treatment when the primary lesion is first recognised. The disease has no fatal tendency in the first two stages, but in late syphilis the gumma may act like other tumours, and cause death by direct interference with function, especially in the brain and meninges. Death also results from syphilitic disease of the arteries, from bronchial, tracheal, or rectal stenosis, from periostitis with necrosis of bone and pyæmia, from lardaceous disease of the liver, spleen and kidneys, and from the parasyphilitic disorders.

Diagnosis.—The spirochætæ of syphilis may be detected in the serum of recent lesions. They may be coloured by Giemsa's stain, or they may be shown by mixing a loop of serum obtained from a lesion with a drop of commercial Indian ink on a microscope slide: the mixture is spread out with the edge of a cover-slip, and soon dries. The spirochætæ are then seen under a high power as bright spirals on a dark brown field.

From a medical point of view it is the recognition of the late syphilitic lesions that is most frequently required, and help is commonly sought in the former history of the patient. The points likely to be remembered are the occurrence of a definite sore other than mere gonorrhœa, the rash, the sore throat and the falling of the hair. Whether the sore was of the hard or soft variety may be unknown to the patient. But since, as we have seen, the poison of syphilis may lie in a soft chancre, the admission that a sore has been contracted, though not proving syphilis, still leaves it possible. The patient may be able to give consistent accounts of the rash, or of the ulcerated sore throat. In married women much reliance is often placed upon the previous occurrence of miscarriages; but miscarriages are frequent under quite different circumstances, and one miscarriage, from whatever cause, is liable to be followed by others.

Search should be made for scars of the original sores on the penis in men, for scars of tertiary lesions on the skin and in the throat, for nodes on the tibiæ and skull, for hardness or atrophy of the testes, and for evidences of lardaceous disease, in the size of the liver and spleen, and in the existence of albuminuria. In earlier lesions the condition of the posterior cervical glands should be noted.

The diagnosis is sometimes assisted or confirmed by therapeutic measures: when, for instance, a suspected lesion yields rapidly to the treatment mentioned below. But this is not applicable in parasyphilitic affections which are less readily benefited by anti-syphilitic drugs.

By bacteriological methods it is now possible to detect in the

blood serum a specific result of syphilitic infection: it is known as *Wassermann's reaction*. The test depends upon the bacteriological conditions which determine *hæmolysis*. It has been previously shown (*see* p. 20) that the repeated injection of the blood of one animal (*e.g.*, ox) into another animal (*e.g.*, rabbit) leads to the formation of an immune body in the latter's serum, and that such serum will then cause the destruction of the blood-corpuscles of any animal of the same species as the former; that such destruction is in part dependent upon the presence of *complement* (or *cytase*) in the serum, which can, however, be destroyed by a temperature of 56° C., and that if it is so removed the corpuscles are not destroyed by the serum. Now if the anti-body of syphilis, such as may be contained in the serum or cerebro-spinal fluid of an old syphilitic or parasyphilitic patient, is mixed with a substance containing the *antigen* of syphilis (*i.e.*, the substance which has the power of calling into existence the anti-body), such as the liver of a syphilitic fœtus, that combination has the power of combining with, or neutralising, complement in such a way as to prevent it from combining with any other mixture of anti-body and antigen subsequently added. For the test the suspected serum believed to contain syphilitic anti-body is mixed with the syphilitic liver, and with normal guinea-pig's serum containing complement. These are kept for an hour at a temperature of 37° C., and the mixture of rabbit's serum containing *amboceptor* and the washed ox-corpuscles is then added, and the mixture further submitted to 37° C. for two hours. If the serum in question contains anti-body of syphilis, the complement will be bound, or fixed, by it, and the amboceptor in the rabbit's serum not meeting with free complement, will not destroy the corpuscles—*i.e.*, there will be no hæmolysis. If, on the other hand, the anti-body of syphilis is not present, the complement will not be bound; it will co-operate with the amboceptor, and hæmolysis will result. The research has to be controlled by similar tests with serum known to be normal, and with serum known to contain anti-body.

Later it was pointed out that human serum contains normally a body which will hæmolyse sheep's corpuscles, and that it also itself contains complement. Hence, since the serum of the patient under trial contains both amboceptor and complement, if sheep's corpuscles be used, the necessity for other sera containing amboceptor and complement is obviated, and the process is much simplified. It has also been found that many other substances will act as the antigen of syphilis; for instance, extracts of normal livers and hearts, some other tissues and organs, solutions of lipoids, lecithin, cholesterin, sodium glycocholate, &c. This last fact, indeed, shows that the substance sought for in the serum is not a truly syphilitic anti-body, as at first supposed, but it is at any rate specific, and in some way the result of syphilitic infection.

The reaction with blood serum is successful in from 70 to 92 per cent. of syphilitic disease, least frequently in primary cases, most

frequently in tertiary cases and parasyphilitic disorders. The cerebro-spinal fluid reacts rarely in syphilis, but with great constancy in the parasyphilitic disorders, locomotor ataxy, and general paralysis of the insane.

Treatment.—Mercury is the drug which has been hitherto universally allowed to have the most powerful influence in the treatment of syphilis. If given with sufficient persistence from the first, it rapidly diminishes the induration of the primary chancre, and it staves off, or considerably modifies, the secondary lesions. Similarly, it lessens the severity and frequency of the outbreaks of tertiary symptoms, if it is continued for some time after the cessation of the secondary lesions. The most desirable method is to give the drug in such doses that it may be continued day after day, and week after week, without inconvenience to the patient—that is, in short, it must not be allowed to cause salivation. It may be given in many forms, but, for the above reasons, the milder are preferable. The more usual are the perchloride in doses of $\frac{1}{16}$ to $\frac{1}{12}$ grain (60 to 80 minims of the liquor), three or four times a day, and hydrargyrum cum cretâ (gray powder) in doses of one or two grains with the same frequency. Hutchinson prefers one grain of gray powder, with a grain of Dover's powder, if necessary; to be given every six, four, three, or two hours.

The method of *inunction* is undoubtedly a very thorough way of introducing mercury into the system, and it is most efficiently carried out at certain health resorts, such as Harrogate, Aix-la-Chapelle, Wiesbaden, and other places. Mercurial ointment, to the extent of thirty or forty grains, mixed with half as much lanoline, is rubbed into the skin by an assistant for from fifteen to thirty minutes. Different parts of the body are selected on successive days; for instance, the arm one day, the forearm the next, the back, chest, thighs, and legs, on successive days, and then the arm again. A complete course includes a daily inunction for six weeks, a rest of three months, another course of six weeks' inunction, a rest of three months, and then a month's inunction, six months' rest, another month's inunction, six months' rest, and finally twenty days' inunction, bringing thus the whole period up to two years.

The *calomel vapour bath*, or *calomel fumigation*, is now rarely employed, as its results are uncertain. The patient sits on a chair, and is wrapped round with blankets, so as to leave the head exposed: 60 grains of calomel are then volatilised by a water-bath over a spirit-lamp placed under the chair. The proceeding lasts fifteen or twenty minutes.

Intramuscular injection is a more recent method of mercurial medication. One-third of a grain of the perchloride is dissolved in 20 minims of water and injected into the gluteus maximus once a week (Bloxam); or one grain of red iodide of mercury in 64 minims of distilled water, with sufficient sodium iodide to dissolve it (dose 2 to 6 minims). Calomel can also be employed in the same way, one grain being suspended in sterilised olive oil: and

the metal mercury itself, worked up with lanolin or other fat in the preparation known as *gray oil*, is injected in a dose equivalent to 1 or 1½ grains. Intramuscular injections have been found useful in military practice; but they present disadvantages in the pain of injection, and in the slowness of elimination in the event of an overdose; indeed, a fatal result has occasionally happened.

During the use of mercury the patient should abstain from smoking and from stimulants, frequently clean the teeth and mouth, and live in every way as healthy a life as possible.

The mercurial treatment, whichever form be chosen, should be always, if possible, continued in the primary or secondary stages, and it is frequently of value in tertiary lesions; but here another drug takes the most important position—namely, *iodide of potassium*. Under its use the most serious and alarming nervous symptoms, due to syphilitic lesions, rapidly subside, ulcerating skin lesions quickly heal, and syphilitic bone-pains and nodes disappear. Five or seven grains three times a day are often sufficient, but in serious cases it may require to be pushed to ½-drachm or drachm doses three times daily; or, in a still worse case, 20 grains may be given in a little milk every two hours through the whole day and night. The advantage of this, no doubt, lies in the thorough saturation of the system; otherwise, as the salt passes away rapidly by the kidneys, the amount in the body may fall very low in a long night interval. If iodide, in any dose, causes coryza, it should be taken much diluted—*e.g.*, in half a tumblerful of water; arsenic may be added if it causes eruptions (*see* Medicinal Eruptions). General tonics, good food, and sea air are desirable if it causes much depression, or the iodide of sodium may be given instead in corresponding doses, or a mixture of the iodides of potassium, sodium, and ammonium in equal parts. If these fail, recourse may be had to mercury, either alone or with a tolerable dose of potassium iodide.

Arsenic has long been thought to have an influence in syphilis, and Donovan's solution, the liquor arsenii et hydrargyri iodidi, was much used by older practitioners. More recently atoxyl and soamine (*see* p. 103) have been tried, but as on more than one occasion blindness has been produced, they cannot be recommended. A still newer arsenical compound is dioxy-diamido-arseno-benzol, which is known as "606," or Ehrlich-Hata. Half a gramme (7½ gr.) of the sodium salt of this drug is injected subcutaneously, or into muscle, or into vein; and remarkable results have undoubtedly been obtained in the rapid healing of syphilitic lesions. It is interesting that the Wassermann positive reaction is not as a rule converted into a negative one, and the frequency of relapses and of toxic effects, which may certainly be fatal, have yet to be learnt. (*See also* p. 41.)

The destruction of the primary sore is probably useless to prevent infection. To soft sores iodoform is a good application, or healthy action may be set up by the use of the acid nitrate of mercury. Iodoform is also of value as ointment or powder to

ulcerating skin lesions, and by means of insufflation to ulcerating tonsils.

Prevention.—As syphilis is rarely conveyed otherwise than by direct contact, it should be easy to prevent its spread, if those who are known to be infected could be made to abstain from contact, sexual or otherwise, with healthy persons. There are many difficulties in the way of legislation. The following are some of the facts which may guide the medical man in advising patients. Both primary and second lesions are contagious, and the blood, during these periods, contains the virus. On the other hand, it seems clear that the normal secretions—saliva, milk, sweat, and semen—do not contain the virus in such a form that it can be inoculated into abrasions, though it will be seen that by means of the semen the fœtus may be infected most completely. The tertiary lesions are not contagious, and probably the blood at that stage is innocuous to other persons (*see* also below).

As a rule, syphilis confers upon the sufferer immunity from fresh infection; but instances are recorded in which, after a long interval, a fresh primary sore and fresh secondaries have appeared, and here we must suppose that the protective influence has died out, as it does rarely in the exanthems.

CONGENITAL SYPHILIS

Children born of parents suffering from syphilis in the first or second stages may themselves be infected with the disease. As a rule, this transmission does not take place in the tertiary stage. The disease may be taken from the father alone, the mother being healthy (sperm-inheritance, or paternal conception inheritance); or from the mother alone, the father being healthy (germ-inheritance, or maternal conception inheritance); or from both father and mother; or, lastly, the mother may acquire syphilis after conception, and convey it to the fœtus *in utero* through the placental blood (pregnancy inheritance). According to Hutchinson, the symptoms of the child's disease are the same whether the disease has been acquired in one or other of these ways; and whether the disease of the parent was in the primary or secondary stage. And the symptoms are not necessarily more severe when they are derived from both parents than when they come from one alone. Further, Hutchinson denies the universality of the law by which the children born at the earliest period of the parent's disease are more severely affected than those born afterwards. As a fact, the transmission is irregular—one child may be badly affected, another not at all. Experience of congenital syphilis has, however, brought out a law to which the exceptions are very rare—namely, that if a child inherits syphilis from its father, the mother being originally healthy, the syphilitic infant may infect a wet nurse—*e.g.*, causing a chancre on the nipple—but will not infect its own mother; thereby showing that the mother is in some way protected against

infection, though she may manifest no lesions whatever of a primary or secondary kind. This is called *Colles' law.* But opinions differ as to the extent to which this infection of other persons can take place. It is admitted that it can so happen, if not frequently; and, especially in the case of condylomata or open sores, reasonable care should be taken to prevent it.

Death of the Fœtus.—One effect of syphilis in the parent is the early death of the fœtus, with resulting miscarriage or premature birth—so frequent that the fact of miscarriages having occurred in the history of a married woman may be important evidence as to syphilis in herself or her husband. It is not so easy to say the exact cause of the fœtal death—whether from the immediate effects of the syphilitic virus or from some disease of the placenta. Hard yellow masses have been found in this structure, and some peculiar changes in the villi; but their significance is as yet uncertain. On the other hand, the fœtus not infrequently presents lesions of the bones, viscera, and skin, which show that it may be profoundly diseased. In the bones a change takes place at the line of junction of the epiphysial cartilages and the shaft, allied to that of rickets, and described as osteochondritis. The cartilage may be separated from the bone by soft granulation-tissue or pus.

Early Symptoms.—Such a change is sometimes present in children born alive, the principal epiphyses being separated from their bones, and the limbs consequently lying useless, so as to give the appearance of paralysis. Occasionally also the child is born with a bullous eruption on the skin (*Pemphigus neonatorum*), or the rash comes out very soon after birth. But in a large majority of cases the child is born not only alive, but healthy, fat, and plump, and remains so for three or four weeks after birth. Then it acquires a nasal catarrh, causing the symptom commonly known as *snuffles*, with a discharge—at first thin and serous, afterwards thicker, purulent, and drying up into crusts, which obstruct the nostrils, so that sucking is difficult. At the same time a *rash* appears, most commonly on the buttocks and adjacent parts of the thigh, back, and abdomen. It consists most often of circular patches, brownish-red like the lean of ham, dry, shiny, and inelastic; the patches run together, and form larger areas of irregular shape, but mostly with a well-defined edge. It is not always easy to distinguish this from eczema intertrigo in the situations liable to this last eruption; and the two conditions probably sometimes co-exist. Less frequently the rash is papular, pustular, and bullous. Other lesions occurring in early infancy are stomatitis, ulcerations about the lips and angles of the mouth, rapidly forming cutaneous abscesses, and periostitis. With all this the nutrition of the child may be little affected, but sometimes wasting results, and the face acquires a withered and shrunken appearance like that of an old man. In this stage death may occur; but under treatment, or otherwise, all the symptoms may subside, and the child may show no indications of the taint

for many years, when, often about the time of puberty, symptoms appear which are more or less comparable with those of the third stage of the acquired disease

Later Symptoms.—These are: periostitis, with the formation of nodes; synovitis, especially a chronic synovial effusion into both knees; scaly or lupoid skin eruptions, which are not very common; bilateral deafness coming on with noises in the ears, but without pain or discharge; disseminated choroiditis; iritis and keratitis. The last is common in inherited syphilis—not so in the acquired disease; it causes opacity of the cornea, which gradually increases till the cornea looks like ground glass. It is associated with ciliary congestion, and in late stages vessels may encroach upon the cornea, producing a "salmon patch." Its tendency is to recover. But in addition to these fresh lesions, inherited syphilis may be recognised by some permanent deformities, the result mostly of those changes which took place in infancy. Such persons present a broad forehead, with unusual prominence of the two halves of the frontal bone; the bridge of the nose is broad and sunken; around the mouth are numerous linear cicatrices radiating from the orifice as a centre; and the permanent teeth, as was first pointed out by Hutchinson, show features from which alone an absolute diagnosis of the condition may be made. It is only the upper central incisors that can be relied upon for this purpose, though other teeth may be similarly affected: they are short, narrower at the edge than near the gum, and the edge presents a single central cleft or notch. This notch is at first, soon after the eruption of the tooth, filled by a notched edge of exposed dentine, which soon breaks away. This change in the teeth must be distinguished from the simple transverse marking, which may result from the excessive use of mercury in infancy, causing stomatitis, and interfering with the proper development of the tooth-sacs. Periosteal changes in the tibia may result in a convexity of the anterior border, the so-called *sabre-shaped tibia.* Visceral changes are also not uncommon, such as enlargement of the spleen, cerebral inflammation or degeneration (*see* Cerebral Diplegia), occasionally orchitis, interstitial hepatitis (*see* Cirrhosis of the Liver), anæmia with or without splenic enlargement, and gummata revealing themselves in adult life.

Treatment.—Mercury acts with remarkable rapidity in infantile syphilis. A grain of gray powder three times a day, or liq. hyd. perchlor. 10 or 20 minims, will quickly cure the rash, snuffles, or other symptoms, and improve the nutrition of the child, if it is defective. If these cause purging, unguentum hydrargyri (10 grains) may be rubbed into the palms and soles night and morning, or into the abdomen every night. Iodide of potassium is of less value, but may be given in doses of 2 or 3 grains three times daily. To mucous patches, or ulcerated skin diseases, calomel powder or mercurial ointments may be directly applied.

FRAMBŒSIA

(*Yaws*)

This is a specific contagious disease which is prevalent in tropical countries. It is known as *yaws* in British Colonies, as *pian* in French Colonies, as *puru* in Borneo and the Malay States. It also occurs under different names in South America, West Africa, and the South Sea islands. The disease affects the sexes equally, young people more than others, and dark races more than the white. It has considerable resemblance to syphilis, and is contracted by infection of some superficial lesion, such as a scratch, wound, or insect-bite on the body, mammæ, hands, arms or legs, but rarely on the genital organs. After an incubation of from two to four weeks, during which there is some malaise, the primary lesion occurs at the site of inoculation, and consists of a small papule, which after a week becomes moist with a yellowish secretion, and this dries into a crust. Under this an ulcer forms; it may heal and leave a scar, or develop into a granulomatous mass. The lymphatic glands in the neighbourhood may enlarge and harden, but rarely suppurate. The secondary eruption appears from one to three months after the first appearance of the primary lesion, and is also preceded by some malaise, fever, and pains in muscles, bones, or joints. The lesions begin in the same way. One or more papules appear, which are at first about the size of a pin's head, and increase until they measure from a quarter to two inches in diameter. The epidermis gets thin, gives way, and leaves a raw surface, from which a sero-purulent fluid oozes, and this dries into a crust. In shape the tubercles are round, oval, or irregular from coalescence; soft, not sensitive to touch, but itching. They affect the face, lips, nostrils, neck, arms, axillæ, legs, thighs, palms and soles, buttocks, and vulva. If they improve, they contract, dry up, and leave a discoloration of the skin; but they may form large irregular sores, and lead to deep ulceration, or gangrene.

Arthritis with fever, periostitis, muscular contractures, and neuritis or neuralgia, are conditions which may accompany the eruption. As a rule the disease ends with the shrinking of the granulomata, but there may be a tertiary stage of gumma-like nodules in various tissues, deep ulcers of the skin, or nodes on the bones.

Histologically, the lesions of this disease are granulomata; but the essential cause appears to be a spirillum, the *Treponema pertenue*, which has been found by Castellani in the primary lesions, in the unbroken papules of the general eruption, in the spleen, lymph-glands, and bone marrow, but not in the blood.

The disease has been conveyed to monkeys by inoculation of scrapings from the papules, and of the blood of a patient.

The disease is rarely fatal; and when it is so, it is generally from the supervention of septicæmia or pyæmia.

Treatment.—Cleanliness is essential. Iodide of potassium in full doses, mercury in children, and liq. arsenici et hydrarg. iod. have been found useful internally. Ehrlich's new remedy (*see* p. 145) is also said to be curative. Locally the eruption may be washed daily with solution of perchloride of mercury (1 in 1000), and ulcers may be treated with astringent and antiseptic preparations, such as iodoform and boric acid.

TUBERCULOSIS

By tuberculosis is meant the formation in one or more organs of certain bodies called "*tubercles*," which result from infection by a specific micro-organism—the *bacillus tuberculosis* of Koch.

Tubercle-Bacilli.—These are minute rods, straight, or very slightly curved, measuring 3μ in length and $\cdot5\mu$ in breadth. They have rounded extremities, and present two or more bright spots, often one at each end, which were first regarded as spores. Like other micro-organisms, they can be stained by special reagents, and a method of detecting them by this means in the sputa is described hereafter (*see* Diagnosis of Phthisis).

Some recent observations point to the possibility that tubercle-bacilli are not, as formerly thought, fission fungi, but that they belong to the class of *Streptothrix*, one of the Hyphomycetæ.

Tubercles.—A tubercle in its most typical form is a small firm nodule, which consists of one or more *foci*, having the following structure: Externally *lymphoid* cells, within these *epithelioid* cells, and in the centre a *giant cell*, with several nuclei. Sometimes there is a delicate reticulum or stroma, and invariably the characteristic *bacilli* are present in one or other part, especially in the neighbourhood of the giant cell. Sometimes the giant cells are absent, sometimes also the epithelioid cells, so that the tubercle may consist only of lymphoid cells.

This typical form is shown by the structures known as *gray* or *miliary* tubercles, which have a pearly gray or sometimes yellowish-gray colour, measure from one to two millimetres in diameter, and are well seen in the lungs, liver, and kidneys.

As the tubercle enlarges, it undergoes a process of necrosis either from deficient vascular supply—for no vessel penetrates within the tubercle—or as a result of some chemical substance secreted by the bacilli. It becomes opaque yellow and cheesy in the centre, and the caseous matter, examined under the microscope, shows shrunken leucocytes, fat granules, and *débris*. The caseous centre enlarges, while at its periphery the tubercle may be invading more and more of the organ in which it is situated, the new tubercle becoming cheesy in its turn. In this condition it is known as a *yellow* or *cheesy* tubercle. In the solid organs, large spherical caseous masses are formed, as may be seen in the brain, and to a less extent in the spleen. If the tubercle formed originally on a surface com-

municating with a duct, the centre of the cheesy tubercle may break down, and be discharged, so as to leave a kind of ulcer, which enlarges by a continuance of the same process: this may be seen in the lung, the intestine, and the pelvis of the kidney. Some caseous masses ultimately undergo *calcification* (largely by calcium phosphate), the tubercle-bacilli are destroyed, and the lesion ceases to be infective.

Another way in which tubercle terminates is by a *fibroid change;* chronic inflammation and induration of the surrounding tissue take place, and the tubercle itself shrinks into a fibrous nodule. This is more common on the surface of the pleura and peritoneum, but also takes place in the lungs.

Ætiology.—Tuberculosis is practically an endemic disease: the bacillus is probably wide-spread in all large communities, and the conditions determining its development in otherwise healthy persons are those affecting (1) the virulence of the organism, (2) the susceptibility or resistance of the recipient individual, and (3) the modes of introduction.

(1) Under the first head there is little that can be said but that the *virulence* undoubtedly varies.

(2) In regard to the *condition of the recipient* great importance attaches to hereditary influence: and it is generally admitted that the children of tubercular individuals are more liable than others to tubercular lesions. Statistical observations, it is true, have been hitherto largely confined to pulmonary tubercle, or phthisis. Thus phthisis frequently occurs in the offspring of a phthisical parent, and if both parents are phthisical, or if one has the disease and the other comes of phthisical stock, the tendency on the part of the children is greater. Nevertheless, the phthisical patients who give a history of phthisis in their parents form only about 30 per cent. of the whole number. In extremely rare instances the tubercle-bacilli or their spores are actually transmitted from one of the parents to the fœtus, and this happens certainly in animals. But in nearly all cases what is handed down is a tendency only, and the child is not born with phthisis or any tubercular lesion.

This tendency to suffer from tubercular lesions, pulmonary or otherwise, was known as the tubercular "diathesis," and was believed to be revealed by certain characteristics of physical growth and mental capacity. A dark, beautiful, or tubercular type, and a fair, coarse-skinned, or scrofulous type, were distinguished. But numbers of tuberculous individuals are of quite indifferent build and appearance, who could not be classed with either type; and the most that can be said of the supposed diathesis is that it perhaps represents some delicacy of tissue, which is little resistant to the bacillus.

A disposition to tubercle may be acquired by any circumstance, or combination of circumstances, which seriously lowers the vitality of the body, whether this be a deficient supply of food and fresh air, or prolonged debilitating illnesses, or special toxic influences. The most frequent of these are: (*a*) Overcrowding, and deficient ventilation,

working in close rooms in the fumes of gas, &c.; (*b*) deficient supply of food, which frequently co-operates with the first cause, as well as the next; (*c*) exhausting work, in association with the preceding; (*d*) frequent child bearing in women, and the exhausting drain of lactation; (*e*) exposure to wet and damp; Buchanan showed that amongst communities living on damp and imperfectly drained soils there was an undue proportion of deaths from phthisis and lung diseases; (*f*) enteric fever; (*g*) excessive indulgence in alcoholic drink; (*h*) diabetes mellitus; (*i*) syphilitic cachexia.

In the case of the lungs, there is much to show that inflammatory lesions, there arising, may prepare the soil for the growth of tubercle. It is a common belief that repeated catarrhs, or even one neglected catarrh, will cause phthisis, and though there is little doubt that often the cough, which is the first indication of tubercular deposit, is mistaken for an independent catarrh, prolonged catarrhal bronchitis, or a catarrhal pneumonia, may lower the resistance of the lungs, and so lead, with other favouring circumstances, to tubercular deposit. Antecedents of this kind are, especially, the pulmonary inflammations following measles and whooping-cough, croupous pneumonia occasionally, pleurisy, and the bronchial irritation and chronic pneumonia of workers in certain industries.

The *age* of the individual is an important factor. Tubercular invasion is especially common in young persons: in infants and young children the meninges, peritoneum, lymph-glands, bones, and joints are attacked; in young adults pulmonary tubercle is common. Persons over forty are much less commonly affected for the first time with tubercle, though tubercular lesions may persist in them up to the ages of fifty, sixty, or seventy.

(3) Tubercle bacilli may enter the system by *breach of surface of the skin*, by the *respiratory passages*, including the nose and mouth, and by the *alimentary canal*.

The first mode is rare; but in the chronic *post-mortem* lesion, known as *verruca necrogenica*, tubercle-bacilli are found, and tubercular disease of the lungs has resulted in some such cases. Here presumably the person has accidentally infected himself from a patient dead of tubercular disease.

The second has long been accepted as the most probable explanation of a large number of cases of pulmonary tubercle, and many cases of tuberculous cervical glands. Not that it suffices to come into occasional contact with, or breathe the air exhaled by, phthisical patients; for the infrequency of bacilli in such air has been shown, and the rarity of such contagion is common experience. In support of this the immunity of the resident medical officers and nurses of the Brompton Hospital for Consumption has often been quoted, and this is confirmed by the experience of the Mount Vernon Hospital for fifteen years, recently recorded by Dr. Squire. But the disease is occasionally transmitted from husband to wife, or has passed between brothers and sisters, or others living together. The researches of Cornet showed that the chief agent in the diffusion

of the bacillus was not the air expired by the phthisical patient, but the sputum, which, as is well known, may be loaded with the specific micro-organisms. If this is repeatedly ejected on to the floor of a room and allowed to dry, or if quantities of it dry upon handkerchiefs, the air of the room may at length be sufficiently impregnated to become dangerous to healthy people breathing it. From the floor and walls of rooms formerly tenanted by phthisical people, Cornet obtained bacilli, by the inoculation of which he produced tubercular disease in healthy animals. This helps to explain the deadly influence of deficient ventilation in workshops, manufactories, barracks, and similar institutions, as well as some of the instances of fatal prevalence in families. Attention has been called to the occurrence of tubercle in birds kept as domestic pets, such as canaries and parrots, and the possibility of infection from them.

Infection by the *alimentary canal* implies the ingestion of food which is tuberculous. It is well known that cattle suffer from a form of tubercle, and that the bacilli differ in certain particulars from those common in human tuberculosis; but, in spite of the view expressed some years ago by Koch that the bovine disease could not be transmitted to man, it is definitely proved now that such transference can take place, and, indeed, it is shown that from 10 to 20 per cent. of bacilli cultivated from human tubercles are of the bovine type. The prevalence of abdominal tuberculosis among children suggests infection of this part of the body by milk, which forms so large a part of the diet of the young. Experiments, however, show some curious facts in connection with the absorption of tubercle. Thus it has been shown that tubercle-bacilli injected into the alimentary canal of young animals passed through the intestinal wall, and were conveyed to the mesenteric glands, which became diseased; and thence in due course the bacilli invaded the lungs, and the cervical glands. But in adults the bacilli passed the intestinal walls and the mesenteric glands without infecting them, while they quickly produced tuberculosis in the lungs. Similar differences were noted when particles of carbon were injected instead of bacilli. Hence the view has been put forward that not only the abdominal organs, but also the lungs, may become tuberculous through milk infected with bovine bacilli; and even that such a mode of infection of the lungs is more common than that by inspired air.

In the great majority of cases the tubercular invasion is *localised* at first to one part, and only slowly spreads either by extension in the tissue it invades, or by means of the lymphatics to the glands beyond.

A *general infection*, in which tubercle appears at the same time in several organs of the body, is nearly always preceded and caused by some local lesion, in the lungs, bones, lymph-glands, genito-urinary organs, or other part. Often there has been no obvious illness, and the patient may be in the enjoyment of good health when the

symptoms first occur; but sometimes, in such cases, caseous bronchial glands or old suppurating foci are found after death. The sudden diffusion of the tubercle-bacillus from the seat of the primary disease to every part of the body is not always readily explained. Tuberculosis of the thoracic duct has been recorded, and the invasion of the pulmonary veins by caseous glands; but these occurrences do not seem to account for most cases, though, obviously, the diffusion must be by means of the veins or lymphatic vessels. Occasionally surgical operations in connection with local tubercular disease may give the opportunity for its spread. Sometimes the disease occurs after measles or whooping-cough, and rarely after typhoid fever; even in these instances a caseous gland or other local tubercular lesion may have existed beforehand.

The results of tubercular disease in each particular organ of the body will be described separately (*see* Tubercular Meningitis, Phthisis, Tubercular Peritonitis, Tubercle of the Kidney, &c. &c.). The following is an account of the disease when it is more widely diffused:

GENERAL OR MILIARY TUBERCULOSIS

The organs which are commonly affected by this multiple growth of tubercle are the lungs and pleuræ, the liver, spleen, kidneys, and membranes of the brain; while less often the choroid of the eye, the heart, thyroid body, marrow of bones, and peritoneum are involved. But the organs in the first group are not all diseased in every case; sometimes one, sometimes another being spared. In any case, the only organs which give rise to definite local symptoms are the lungs and the meninges of the brain; the other symptoms are toxic in kind, and such as are produced by other infectious processes. When cerebral symptoms are present early in the case, they generally mask the pulmonary condition almost entirely, and the case is regarded as one of tubercular meningitis. This disease will be separately described, and it will be noted that after death in such cases a tuberculosis of the lungs and other organs may be found without any clinical warning that they were implicated. If a case of acute tuberculosis is fatal without any cerebral symptoms and without tubercle of the meninges, then the tubercles will be certainly found in the lungs, and of these cases in some the symptoms are obviously pulmonary, while in others they are more general. Thus cases of miliary tuberculosis may be divided into three groups: (1) Those with predominant cerebral symptoms (tubercular meningitis); (2) those with predominant pulmonary symptoms (pulmonary tuberculosis); (3) those with general symptoms of infectious disease, in which also the lungs are usually involved, and which may also present even some cerebral symptoms. It is of the last two forms, in which pulmonary tuberculosis is a part of general tuberculosis, that the present section will deal.

Anatomy.—In miliary tuberculosis one finds the most typical examples of tubercle. Through the lungs the tubercles are, as a rule, uniformly scattered more or less thickly; occasionally only they may show a great preference for the apices. Every form of tubercle may be seen, from the gray, hard granulations, to the larger caseating tubercles; and sometimes these last may be breaking down in the centre, forming minute cavities. Definite patches of pneumonic consolidation occur, but are not common. Some inflammation of the bronchi, especially the smallest, is always present.

Tubercles are sometimes found on the pleuræ, and pleurisy is often the result. In cases grafted on a former phthisis, consolidation and cavities will also be present. In the other organs mentioned tubercles are also found, of different ages in different cases. Tubercle-bacilli have been found in the blood both during life and after death.

Symptoms.—These are at first and often throughout very obscure. The patient complains of weakness, inability to do his work, loss of flesh, anorexia, nausea or sickness, and headache. The bowels may be constipated or occasionally loose for a few days. Pyrexia is present, of a somewhat irregular type, generally high in

FIG. 10

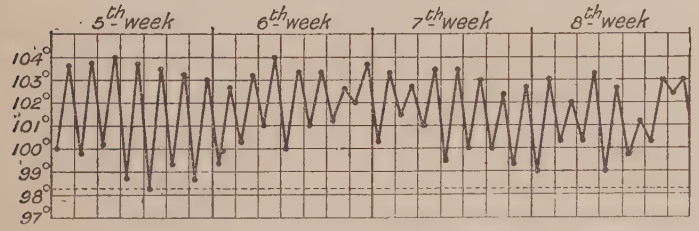


Chart of a Case of General Tuberculosis, fatal at the beginning of the Tenth Week.

the evening and much lower in the morning; thus the evening temperature may range from 100° to 103°, the morning temperature from 98° to 100°. Sometimes the urine contains a trace of albumin. Physical examination in other parts may reveal nothing; but the patient gets more emaciated, the pulse is rapid and feeble, the tongue is dry, food is taken badly, and prostration becomes marked. Pulmonary symptoms may then become more prominent, such as cough and scanty mucous expectoration; and on auscultation more or less extensively diffused râles may be heard. Or some cerebral symptoms may manifest themselves, such as strabismus, unilateral ptosis, weakness of one leg, twitching in one or other limb, or general convulsions. The prostration increases, there may be coma, and death is the result. Very rarely jaundice is present. The duration is from four to ten or twelve weeks.

In other cases the pulmonary symptoms are from the first more

marked. Here also the loss of strength and emaciation are pronounced, but quite early there occur cough, dyspnœa, scanty mucous expectoration, tinged, it may be, with blood, and sometimes pain in the side. The physical signs are at first suggestive of bronchitis. Resonance is but little affected, there may be a slight impairment at one apex; or, on the other hand, some increase of resonance over the whole chest. With the stethoscope one hears sibilant and sonorous rhonchi, fine and small râles, of which many are consonating. Only occasionally one gets scattered patches of high-pitched breathing or obscure dulness. If the condition is secondary to an old phthisis the signs of this will, of course, be observed at the same time. When these conditions are well marked the patient presents a high degree of cyanosis—the face, lips, nose, ears, and cheeks being livid, and the fingers shrunken and blue. The temperature has the characteristics already noted. Sometimes the *typus inversus* is present, the morning temperature being high and the evening low.

Death at length takes place, after from three to eight or ten weeks, with increasing dyspnœa, lividity, prostration, and drowsiness.

Diagnosis.—The insidious beginning, and the comparatively rapid prostration with a febrile illness, easily lead one to confound this disease with enteric fever, and the symptoms of bronchitis, as long as they are moderate, rather increase the difficulty. The points in favour of enteric fever are rose spots, typical diarrhœa, distended abdomen, a successful Widal reaction (*see* p. 52), and leucopenia; those in favour of miliary tuberculosis are rapid emaciation from the first, the mixed pallor and cyanosis of the face, as contrasted with the pink flushed cheek and white face of enteric fever, the rapidity of breathing out of proportion to the other signs of illness, and a normal number of leucocytes. The occurrence of any cerebral symptom should at once give the clue to the nature of the disease. The fundus of the eye should be examined for tubercles, though they are present in only a minority of cases; optic neuritis is less likely to give assistance, since it is not generally seen before the onset of the cerebral symptoms themselves, and, moreover, occasionally occurs in enteric fever. The pronounced pulmonary cases are more likely to be confounded with bronchitis or broncho-pneumonia. High fever, rapid emaciation, and marked cyanosis distinguish acute tuberculosis from simple bronchitis. Cases of broncho-pneumonia present greater difficulties; the physical signs, the remittent pyrexia, and some cyanosis are common to both. Generally, the shorter duration, or the presence of rather decided areas of consolidation, will point to broncho-pneumonia; but broncho-pneumonia may last long enough to be mistaken for tuberculosis. When a child has pulmonary complications after whooping-cough it is often very difficult to distinguish between broncho-pneumonia and tuberculosis; in this case it is more common to diagnose the former disease or to overlook the fact that the latter may be there. It

may be suspected if the symptoms are prolonged for several weeks with increasing cyanosis and wasting. In any case the ophthalmic or cutaneous tuberculin test may be applied (*see* Diagnosis of Phthisis).

Prognosis.—Though it is believed by some that miliary tuberculosis may recover, the cases must be rare, and the tendency would be, in the absence of unequivocal evidence of tubercle (bacilli in blood or sputum), to believe that the case had been either enteric fever or broncho-pneumonia.

Treatment.—A case of developed general tuberculosis is rarely amenable to the forms of hygienic treatment which are often successful in the localised forms of the disease (*see* Phthisis, Tubercular Peritonitis, &c.). The patient is confined to his bed, and requires constant medical attendance and nursing. Specific treatment by the injection of tuberculin is little likely to have any effect. Otherwise the treatment must be purely symptomatic and supporting. Abundant fluid nourishment and small doses of stimulants should be given; opiates may be administered to relieve pain or distressing cough, and ammonia to act as an expectorant.

LEPROSY

(*Lepra. Elephantiasis Græcorum*)

A chronic infectious disease characterised by nodular lesions of the skin, mucous membranes, and nerve-trunks. Bacilli (*B. lepræ*), first described by Hansen, are constantly present in the affected tissues. They closely resemble tubercle-bacilli, but differ in some colour reactions, and in always being straight. Up to the present no medium is known upon which they can certainly be cultivated, and inoculation of animals has been unsuccessful, except in the case of monkeys, in which a temporary local infection has been obtained. In man the bacilli have been found in the skin and mucous membranes, in the nerve-trunks, in lymph-glands, in the larynx, liver, spleen, testes, kidneys, and rarely in the lungs; but not in the muscles, bones, and joints.

Ætiology.—The disease is not common in Europe, except in Norway, and is, as a rule, only seen in England in the case of patients who have lived in the East or West Indies. But it is found in some parts of South Europe, and, amongst other places, in India, Burmah, Siam, China, Japan, the North-East of Africa, the Cape of Good Hope, the West Indies, Mexico, Central America, and parts of South America, and many islands of the Pacific. It is, however, not peculiar to one kind of climate or soil. Males are more often affected than females, and the disease is commonly contracted in early life, before the age of thirty, and rarely in infancy. But it is not congenital, and heredity seems to have but little share in its occurrence. The firm belief that it is contagious has for centuries influenced the customs of the countries in which

it is prevalent; yet direct transmission has only occasionally been proved, and nurses have lived in leper institutions for years with impunity. It has, however, been successfully inoculated in man, and its spread in new countries after the arrival and settlement of a leprous person has been observed even in modern times, as in New Caledonia and Mauritius.

The entrance of the bacilli is probably effected in different cases by one or other of the following openings: the nasal and upper respiratory passages, the mouth and tonsils, the abraded skin, or the genital organs.

Symptoms and Course.—The disease often begins with some general indisposition or malaise, with slight fever; and the lesion of the skin first shows itself as red or brownish-red spots on the limbs or trunk (*Lepra maculosa*). They may be from half an inch to three or four inches in diameter, are round or irregular in shape, and slightly swollen. Sometimes they form rings by clearing up of some spots at their centres. With the subsidence of the pyrexia they may also fade, but are apt to leave pigmented stains, or sometimes white spots, behind them; and from time to time fresh illnesses and fresh outbreaks of spots occur.

In the form known as *Lepra tuberculosa* or *L. nodosa* there occur, simultaneously with the spots, or after an interval, hard elevations of the skin, from the size of a pea to that of a hazel-nut or larger. They may persist for a long time, and may ultimately disappear, leaving pigment spots behind; or they may soften, break through the skin, and leave indolent ulcers, having weak granulations, and discharging a scanty thin pus. The nodules appear mostly on the face, on the dorsal surfaces of the hands and feet, and on other parts of the limbs. On the face they are apt to produce great deformity by the enormous thickening of the eyebrows, the nose, cheeks, and lobes of the ears; and the very characteristic appearance thus produced is described as *leontiasis*, from its resemblance to the face of a lion. The tubercles also develop in the mucous membranes of the mouth, the gums, the palate, the larynx, or the nose; and the voice may be rough and hoarse, or high-pitched and feeble, in consequence. The ulcers may eat deeply into the parts beneath, so as to erode tendons and bones, and open the joints.

In *Lepra anæsthetica* nervous symptoms predominate, but they often co-exist with the conditions above described. They consist partly of tingling, partly of numbness and actual anæsthesia, as proved by the inability to feel the prick of a needle-point. This is found in various isolated patches on the limbs and trunk; and over these patches the skin is either paler than normal or more pigmented, the hairs are small and wanting in pigment, and the skin generally is smooth and glistening. In this form, also, deep ulcerations take place over the joints of the fingers and toes, and the phalanges, or carpal or tarsal bones, may ultimately be shed; the terminal phalanges, it is said, often being spared, and the

wounds sometimes healing up with remarkable completeness (*Lepra mutilans*).

With remissions and exacerbations the disease has a hopeless course, rendering the sufferer a loathsome object to look at, but not for a long time depriving him of appetite, or otherwise interfering with the performance of vital functions. Death takes place after five, ten or fifteen years, from tuberculosis of the lungs, nephritis, or other intercurrent affection; and occasionally from obstruction of the larynx, gangrene, or pyæmia, the more direct results of the leprosy.

Anatomy.—The nodules are in the skin beneath the epidermis, and processes descend into the subcutaneous tissue. They consist of granulomatous tissue containing *lepra cells*, which are cells containing vacuoles, one or two nuclei, and bacilli in great numbers, singly and in clumps. The cells vary in size from that of a leucocyte to three or four times as large, and are at first grouped round the blood-vessels. In anæsthetic leprosy the nerves are the seat of neuritis. They are often thickened to two or three times their normal size, from proliferation in the sheath, by which finally the axis-cylinders may be atrophied or destroyed. Nodular infiltrations also form, which can be felt under the skin in the case of superficially-placed nerve-trunks.

Diagnosis.—There is generally little difficulty in the recognition of the developed disease. If necessary, the bacilli can be sought in the serum of a nodule, or in the pus from an ulcer.

Treatment.—The disease is practically incurable. The most that can be done is to remove the patient to a locality in which the disease is not endemic, to give good food and cod-liver oil, and apply simple dressings to such ulcers as may be present. Chaulmoogra oil, in doses increasing from 20 minims to 2 drachms in the day, and antileprol, a purified form of the same, in doses of 4 to 15 grains in capsule, are believed to do good. Serum therapy has not as yet produced any results. Deycke isolated from leprous nodules a streptothrix, and from this a neutral fat, *nastin*, which he found to have a bacteriolytic effect on the lepra bacilli. He has used injections of nastin with benzoyl-chloride, and claims to have obtained promising results. Other observers have been less successful.

SEPTICÆMIA, SAPRÆMIA, AND PYÆMIA

The process of suppuration is caused by a number of organisms, of which the *staphylococcus pyogenes aureus* and *albus*, and the *streptococcus pyogenes* are the most frequent. Constantly present, and constantly brought into contact with the skin and mucous surfaces, they enter through cuts, fissures, and abrasions, and produce effects which vary with their virulence, with their numbers, and with the resisting power of the tissues and fluids of the body. Their local effects are the production of inflammation and

suppuration, of which the latter is the liquefaction of the tissue with accumulation of numberless leucocytes, attracted in the process of *chemiotaxis*. These local lesions on the surface of the skin are known as *pustules, boils, furuncles,* or *carbuncles;* in the deeper parts, as *cellulitis* and *abscess;* and on mucous membranes as *catarrh*. An allied condition is *erysipelas,* which will be described separately.

When the lesions are of slight extent there may be no appreciable fever or other general result; that is, the absorption of toxins may be extremely slight, or they have no marked virulence.

Far more often they are accompanied by fever, which is an index of the operation of toxins after their absorption. In this case there is *intoxication* or *toxæmia,* the organisms being confined to the seat of the lesion, and not necessarily or at first entering the body.

A general infection by the organisms is a further development of a most serious kind; this is effected by their reaching the circulation and being carried thereby throughout the body. Pyogenic organisms are rarely found in large numbers in the superficial blood-vessels, but are very numerous in the capillaries of the viscera. This condition is known as *septicæmia,* and is distinguished from the preceding by the fact that in the former the cure of the local condition removes the source of the toxins, and hence the toxæmia must cease, when the toxins first absorbed have been eliminated. In the latter the organisms, being diffused in the blood, continue to produce toxins, in spite of adequate treatment of the local disease.

Pyæmia is a still more dangerous condition, in which the minute blood-vessels in different parts of the body are blocked by fragments of thrombus, pus, or *débris* containing micro-organisms, so that minute abscesses form in the viscera, from which further absorption of toxins takes place, so as to render a fatal result almost inevitable.

The last three conditions are characterised by febrile reaction of different degrees of intensity, analogous to what occurs in other infectious disorders (typhus, typhoid, pneumonia, or diphtheria): and so nearly or so often identical that the greatest difficulty may be experienced in distinguishing between them.

SEPTICÆMIA

This may arise in any individual from infection through small wounds, or injuries by dirty tools or instruments; or after large wounds from surgical operations or from accident; or from the uterus after parturition or miscarriage. A rise of temperature is one of the earliest signs, and this may be accompanied by a rigor. With this the tongue is furred, and there are anorexia, perhaps vomiting, prostration, weakness, and finally all the conditions of the typhoid state, such as delirium, stupor, dry brown tongue, dusky or sallow complexion, and tremor of the limbs. Sometimes there are loose motions, and sometimes patches of erythema on the skin, or petechiæ. The duration of the symptoms is variable, and

may be from two or three days to eight or ten in fatal cases. Milder cases may recover after a much longer period. After death the conditions are found which have been described on page 26.

Treatment.—The wound should be treated as promptly as possible on strictly antiseptic methods. The diet of the patient must be that employed in the severe forms of fever, such as typhus, typhoid fever, and scarlatina; and stimulants are likely to be required early. Success has followed the use of antistreptococcus serum in some instances; it is, of course, more probable in cases definitely due to streptococcus infection. If possible, a serum prepared from a variety of the organism corresponding to that infecting the patient should be employed: otherwise a polyvalent antistreptococcus serum may be tried. Vaccines, with the assistance of the opsonic index, may also be employed.

SAPRÆMIA

In this condition the general symptoms are identical with those of septicæmia. They are due to the circulation in the blood of poisonous substances (toxins or ptomaines), which result from the action of putrefactive bacteria upon necrosed tissues; and sapræmia differs from septicæmia in the fact that the organisms themselves are confined to the local lesion, and do not penetrate into the blood. Efficient local treatment by removal of the source of the toxins is promptly followed by improvement; for no more toxins are poured into the blood, and those already there are eliminated in the urine, or otherwise.

PYÆMIA

The formation of abscesses in various parts or organs, which distinguishes pyæmia, may be the consequence of an open wound, accidental or operative, or of a collection of pus, in any part of the body. Pyæmia was in former times the scourge of the surgical wards of a hospital, until the almost universal use of the antiseptic methods of treatment introduced by Lord Lister. It may, however, arise from lesions which come frequently under the notice of the physician, such as ulcerations of the mucous surfaces, and which are not amenable to antiseptic treatment; and it occurs exceptionally without any preceding lesion, so far as can be ascertained by the most careful examination, whether during life or after death (so-called *idiopathic pyæmia*).

The name pyæmia (pus in the blood) arose from the idea that pus was actually transferred from the original lesion to the seat of the secondary abscesses along veins in which no protective coagulum had been formed. Abscesses, which are the distinguishing features of this disease, are formed as the result of infarction or embolism (*see* Embolism) of minute vessels, with portions of thrombus or *débris* carrying the infective organisms; so that not only obstruction

of the vessels, but also inflammation and suppuration, occur. The organism is in most cases the *streptococcus pyogenes,* less commonly a *staphylococcus.* The thrombus is frequently provided by the coagulation of blood in the veins in connection with the wound. Embolism is most common in the bases of the lungs, where the wedge-shaped or conical areas of lobular pneumonia and the resulting abscesses are the most characteristic feature of pyæmia. But abscesses occur in other parts of the body, such as the liver, spleen, and kidneys, which are not within the pulmonary circulation. Cocci are found in the secondary lesions, both in the capillaries and in the tissues; and they are seen, but not constantly, in the blood. The occurrence of abscesses in the range of the systemic circulation is not very easily explained. The embolic particles are not likely to pass through the capillaries of the lung, and it must be supposed that cocci which can so pass may become aggregated together to form emboli in the liver, spleen, and kidneys; or that the organisms are deposited in the endothelium of vessels and there grow, or perforate into the tissues and form abscesses.

Secondary abscesses, indeed, may occur in nearly every part of the body, but particular organs are associated together in ways that are partly, but not altogether, explained by the course of the blood-stream. (1) In the more common *acute fatal pyæmia,* the abscesses are nearly always found in the lung, and perhaps there alone. They are often associated with acute pleurisy, which may be serous or purulent; this is mostly referable to the proximity of the abscesses to the pleural membrane. Pericarditis and peritonitis also occur. (2) Another kind of case is that which begins in an *acute infective osteomyelitis* (*acute necrosis*): here the secondary abscesses form especially in the cardiac muscle and in the kidneys. (3) In *portal pyæmia* the primary lesion is some form of ulceration of the parts which drain their blood into the portal vein; and secondary abscesses form in the liver, with or without a suppurative pylephlebitis (*see* Pylephlebitis). (4) A fourth variety is infective or malignant endocarditis, which was called by Wilks *arterial pyæmia;* and in which micro-organisms and *débris* of thrombus are conveyed in the blood-stream to distant organs, and may produce suppurating infarcts, especially in the brain, kidneys, and spleen. This closely resembles ordinary pyæmia; indeed, it may form part of the pyæmia arising from an external wound (*see* Malignant Endocarditis).

In *chronic pyæmia* the viscera are mostly spared, abscesses form in the subcutaneous tissues, and the joints inflame or suppurate. This is not uncommon in puerperal cases. Watson Cheyne thinks that in these cases the abscesses do not result from emboli, but that organisms circulating in the blood are attracted to regions of depressed vitality, settling probably in the endothelium of the capillaries.

Ætiology.—Apart from accidental and operative wounds, the lesions which lay the body open to pyæmic infection are typhoid or

dysenteric ulceration of the intestine, ulceration of the vermiform appendix (appendicitis), fistula, gonorrhœa, prostatic septic thrombosis, otitis media, and post-partum exposure of the uterine surface. Intemperance, such general conditions as Bright's disease, and acute fevers, are believed to dispose to the occurrence of pyæmia. The fact that so-called idiopathic cases occur is paralleled in other forms of infective disease, such as infective endocarditis and cerebro-spinal fever, where the mode of entry of the organisms is not always apparent.

Symptoms.—The disease often begins suddenly with a prolonged rigor, followed by profuse sweating and collapse, the temperature rises, and fever continues to be interrupted by fresh rigors daily, or two or three in the day, but often without any regularity. There are anorexia, thirst, and dry tongue; anxiety, prostration, rapid breathing, and loss of flesh. The face is usually sallow, or even distinctly jaundiced, and the urine may contain some bile-pigment. Sickness is not infrequent, and diarrhœa may be present. Leucocytosis is marked. The rigors may cease after five or six days, but fever of an intermittent or remittent type continues; occasionally there are transient erythematous patches in various parts of the body. As already stated, the local lesions vary, and the symptoms differ accordingly. When the lungs—as is common—are the seat of secondary abscesses, the respirations are rapid, with supplementary breathing in front, deficient entry of air at the bases, and perhaps sharp cracking râles; or there are dulness, tubular breathing, and other signs of pulmonary consolidation or pleural fluid. Pericarditis or peritonitis will be shown by its characteristic symptoms. The duration of these cases is often quite short—from six to ten days; a typhoid condition ensues, with prostration, stupor, delirium, dry brown tongue, quick feeble pulse, and death.

In the *chronic* cases, where the viscera are spared, and the abscesses form in the joints, the latter become swollen, tender, and hot; tender points appear on the surface of the limbs or body, and beneath them abscesses rapidly form, with thin, unhealthy pus and imperfectly developed limiting walls. Fresh abscesses occur from time to time for several weeks or months, and the patient may ultimately recover, sometimes with ankylosis of joints; or death may take place from persistent toxæmia. The symptoms in other cases may be modified by the special localisation of the secondary lesions. In pyæmia secondary to otitis the lungs are implicated, or there may be pleurisy or empyema with the substance of the lung nearly free. If meningitis occurs the cerebral symptoms will largely mask the others.

Morbid Anatomy.—In acute cases of pyæmia, the most constant lesions are the abscesses at the bases of the lungs and inflammation of the pleuræ and pericardium; in addition, the blood is dark and usually fluid, the solid organs are softened, and ecchymoses or petechiæ are found under the serous membranes.

Diagnosis.—The occurrence of rigors and profuse sweatings in the course of the treatment of a wound, followed by collapse and a typhoid condition, while the wound takes on an unhealthy appearance, is characteristic of pyæmia. Where these symptoms occur without any external wound the same diagnosis may be obvious; and search will have to be made after the primary lesion, which may prove to be otitis with discharge from the ear, disease of the nose, intestinal ulceration, or abdominal suppuration. In the last two cases, the lesions are probably confined to the portal circulation (*see* Suppurative Pylephlebitis). Sometimes the rigors take place with such regularity as to resemble *malaria;* resistance to quinine, a causative lesion, and the absence of malarial parasites from the blood would be in favour of pyæmia. *Infective endocarditis* is generally distinguished by the presence of a cardiac murmur; but endocarditis of the pulmonary valves may be caused by pyæmia secondary to a suppurative lesion.

In the late stages pyæmia may closely resemble *enteric fever;* especially if there is no discoverable lesion to suggest the former: rigors, however, are uncommon in enteric fever. Lastly, joint pains, like those of *rheumatism,* within a few weeks of confinement or miscarriage, should always excite a suspicion of pyæmia. In this disease the inflammation persists in each joint as it is involved; whereas in rheumatism the pains often shift from joint to joint, and may return again in those first affected.

Treatment.—This is almost hopeless in the visceral forms; but less unpromising in those with synovitis and cutaneous abscesses. The injection of a polyvalent antistreptococcic serum has been of great use in many cases, and should certainly be tried. Failing this, or at the same time, quinine (5 grains) or sodium sulphocarbolate (10 grains) may be given every four hours; and in any case nourishment and stimulants must be supplied freely. If the primary lesion can be reached, it should be dealt with surgically, so as to get free drainage and asepsis; and secondary abscesses should be opened where accessible.

ERYSIPELAS

(*St. Anthony's Fire. The Rose*)

Erysipelas is a specific contagious disease, characterised by a peculiar form of inflammation of the skin, and due to the invasion of the *streptococcus pyogenes* or *erysipelatos.*

Ætiology.—The most common determining cause of erysipelas is the presence of a wound, whether accidental or the result of operation; and infection, no doubt, takes place through this breach of surface, and spreads to the surrounding skin. Even though it sometimes arises, apparently, without any wound, it will in such cases generally be found that there is a slight scratch or an abraded pimple, or other very slight lesion of the skin.

Though thus contagious and inoculable, the infection is active only over short distances.

It affects infants and people over forty years of age more frequently than others; men and women are about equally prone to it. Some conditions of the individual increase the liability:—chronic disease of the liver and kidneys, chronic alcoholism, and malnutrition from insufficient food. Cold and damp weather, overcrowding, bad ventilation, dirt, and bad food and water may act in the same way. There may be also an individual tendency, for it often recurs in the same person; at any rate, the immunity conferred by it seems to be short-lived.

Symptoms.—Apart from injury and operation, erysipelas most commonly attacks the face, and the present description applies especially to that region. The *incubation* of the disease is probably only a few days—from three to six, or in some instances much longer. The *invasion* is generally by a chill or rigors, and such malaise as commonly accompanies the onset of the specific fevers—headache, anorexia, furred tongue, and general pains. Within a few hours a red, tender spot shows itself on some part of the face, the side of the nose, the inner canthus of the eye, or the external ear. It may be determined by a lesion of the skin if this exists, and it not infrequently begins at the point of junction of the skin with the mucous membrane of one of the orifices—the nose, mouth, or external ear. The spot enlarges, and the skin becomes bright red, swollen, and very tender, and pits slightly on pressure. The inflammation may confine itself to one side of the face, but more often affects both, and may extend to the scalp. It spreads with varying rapidity, the advancing edge is sharply defined, thick, and raised above the surface, and small tongue-like projections can be felt under the skin in front, which is not yet reddened. The whole face may be thus covered in three, four, or five days. At the height of the disease the face presents a remarkable appearance: the features are enormously swollen, of bright or dusky-red colour, the eyelids are distended so as to look like bladders, generally some muco pus is oozing from between them; the ears are thickened and much enlarged, and the patient is absolutely unrecognisable; the scalp is also swollen, and puffy. Often blebs form upon the cheeks or eyelids, which contain yellow sero-purulent or purulent fluid, and these may burst and leave yellow scabs, which further disfigure the patient. The lymphatic glands in the neighbourhood are enlarged and tender, and they are said to be thus affected even before the beginning of obvious inflammation of the skin.

The disease is accompanied by fever, mostly very decided. The temperature generally rises early to 102° or 103°, and reaches a maximum of 104° or 105° on the third or fourth day. About the sixth day it tends to fall rather suddenly, but may remain high if the cutaneous inflammation persists, or may rise again with any fresh outbreak of the local disorder. Indeed, it is closely

dependent upon the inflammation of the skin; in some cases, perhaps more often when the erysipelas is not extensive, the temperature may not rise above 102°. The pulse is quick and full, numbering 100 to 120, or more. The tongue is covered with a thick white fur. The urine is scanty, and in many cases contains some albumin, which may be present for some days. The inflammatory condition invades also the mucous membrane; the palate, fauces, tonsils, and occasionally the laryngeal mucous membrane, may be reddened and swollen, and cause difficulties in respiration and deglutition. The blood shows a condition of leucocytosis. Delirium is common in severe cases, and generally of a low, muttering kind; and coma may follow. While the inflammation is still advancing on one side, it may begin to subside at the points first affected. This receding edge is then less well defined, graduating both in colour and elevation into the healthy skin, as contrasted with the advancing margin. The swelling, tenderness, and pitting on pressure subside in turn over the whole of the affected area; the colour fades somewhat, but mostly changes to a brown tint; and large, thick flakes of dead epidermis now begin to desquamate. This process may take some days. After erysipelas of the scalp, the hair often falls out at the same time as the skin is shed, or somewhat later.

Death takes place from exhaustion, with delirium and coma, especially in older patients, habitual drinkers, and those with chronic visceral disease. It may also occur from complications.

Complications and Sequelæ.—Abscesses may form under the skin, or the tense skin may slough, and induration, or, rarely, suppuration of the lymphatic glands may ensue. The laryngeal œdema may cause asphyxia; pneumonia and pleurisy are occasional complications, and peritonitis and endocarditis have been recorded. Pyæmia and meningitis have been frequently mentioned in connection with erysipelas, but both are rare as direct results of the erysipelas itself. The former may arise from the wound which preceded the specific inflammation; the latter may occur if the original lesion has been a fracture of the skull, or if infection spreads inwards from the orbit. In erysipelas of the scalp, the delirium may be violent or maniacal, and accompanied by delusions, but of itself this is not sufficient to justify a diagnosis of meningitis. Mental disturbance may also be a sequela.

Pathology.—Microscopic examination of the skin of the affected part shows that the cutis and subcutaneous tissues are swollen, œdematous, and filled with large granular leucocytes, which in the upper layers of the cutis closely surround, as well as fill, the lymphatic vessels. The spread of the disease is said to follow the course of the lymphatics, but it is partly dependent on the direction of the connective-tissue meshes, and it is often checked or stopped at lines where the skin is closely adherent to subjacent parts—as, for instance, along Poupart's ligament and the crest of the ilium.

The *streptococcus erysipelatos* is found in the lymphatic vessels and

lymph-spaces at the advancing margin of the disease, as well as in the deeper layers of the skin of the central parts; and rabbits and human beings have been successfully inoculated from its cultivations.

Diagnosis.—Facial erysipelas may be confounded with erythema, acute eczema, herpes zoster, alveolar abscess, and even mumps. Erythema occurs in red patches, generally two or more in number, much less raised, and without pronounced fever. The vesication of an acute eczema and of herpes are distinctive; and herpes is unilateral and confined to one of the areas of distribution of the fifth nerve. M. Milian regards as distinctive of erysipelas the spreading of the redness so that the maximum is always remote from the point of origin, the implication of the ear in the inflammation, and the great tenderness of skin in the advancing zone.

The phlegmonous or cellulo-cutaneous erysipelas, and cellular erysipelas or diffuse cellulitis, described by surgical writers, may be distinguished clinically from cutaneous erysipelas: the first presents more brawny hardness and greater swelling, but no defined edge, and tends rapidly to sloughing; in the second the skin itself is not affected, unless from sloughing of the tissue beneath. But their bacteriological relations are probably not very different.

Prognosis.—Though in most cases favourable, it is dangerous in proportion to the extent of surface involved; and it is often fatal in old patients, and in the subjects of chronic visceral disease, alcoholism, or malnutrition.

Treatment.—The general treatment must be of a stimulant and supporting character. Abundance of milk, beef-tea, mutton-broth, and other fluid forms of nourishing food is required, and in most cases alcohol, in the form of port wine or brandy, must be given. The tincture of ferric chloride used to be regarded as a specific for erysipelas; it at any rate acts as a good tonic, and should be given in doses (for an adult) of 30 to 40 minims every three or four hours. Quinine has also been recommended. Good results have sometimes followed the use of an *antistreptococcus serum*, injected subcutaneously in doses of 15 c.c. or 20 c.c. once or twice daily. This serum is obtained from the horse after the animal has been immunised by repeated inoculation with streptococcus (*see* Diphtheria antitoxin, p. 125). Tepid or cold sponging may be resorted to where the fever is unusually prolonged or high. Locally, relief is given by dusting the face or other part affected with a powder of starch, zinc oxide, or boric acid; or by applying an ointment of vaseline with 10 or 15 per cent. of ichthyol: elsewhere than on the face this may be covered with a layer of cotton wool or wadding. Lint soaked in lead lotion, or lead and opium lotion, will also give relief, but should not be used where there is any tendency to gangrene. If there is extreme tension of the skin, a few small incisions may be made in it; but there is often a very deceptive sense of fluctuation, even though pus is not present.

RHEUMATIC FEVER

(*Acute Rheumatism*)

Rheumatic fever is a febrile disease in which there is acute inflammation either of the joints, or of the heart and its membranes, or of both together.* There can be little doubt that it is due to a micro-organism; and this has been described as a diplococcus by Poynton and Paine, and as a streptococcus occurring in pairs or short chains by Ainley Walker. It has been isolated in fatal cases from the blood, cardiac valves, pericardium, and tonsils; it can be cultivated on artificial media; and lesions resembling those of rheumatism have been produced in animals by inoculation of cultures.

Ætiology.—The disease occurs in both sexes, and at nearly every age; but it is very rare after fifty years of age, and in infants. It occurs with greatest frequency in its articular form in adults between fifteen and thirty, and quite commonly, though in a somewhat different form, in young children. The tendency to its occurrence is by many thought to be hereditary. Cold and damp are regarded as exciting causes, and appear to be in operation in a large proportion of the cases. It is no doubt in accordance with this that it is least frequent in England in the months of July, August, and September, and that the sufferers from rheumatism include a large proportion of servant girls and others of the poorer classes. Rheumatism has important relations to chorea, which will be referred to later; and scarlatina may be followed by a multiple synovitis, which, if sometimes certainly septicæmic, is at others quite indistinguishable from acute rheumatism.

Symptoms. *Arthritis.*—The onset of rheumatic fever is sometimes quite sudden, so that the patient first feels a pain in one joint and then successively in others; or there are a few days of obscure illness before the pains in the joints occur to mark its nature. There may be a little sense of chilliness, but there is rarely a distinct rigor such as occurs in pneumonia or pleurisy. The knee is often first attacked, and then the ankle; in other cases the wrist or the shoulder. Whichever is first attacked, the disease may soon spread to other joints of the body, so that the shoulder, elbow, wrist, and phalangeal joints, the hip, knee, ankle, and phalangeal joints of the toes, may all be inflamed at the same time or successively. Not infrequently the sterno-clavicular joint, and even the vertebral and costo-vertebral joints, are undoubtedly affected. But the extent of the disease is very variable. In one only two or three joints may be inflamed, in another a great number; and an important feature of rheumatic fever is the way in which some

* The terms rheumatism and rheumatic have been both popularly and in medical works used with little discrimination for many disorders in which *joints* or *pains* or *cold* are concerned; the last perhaps more correctly, for the name is derived from ῥέω, I flow, and is associated with the word catarrh, καταρρέω. But it is very desirable that the terms should be restricted to the disease now being described, and to conditions presumably allied to it.

inflamed joints will quickly recover, while others become involved; and these last will get well, while fresh joints suffer, or those first affected become again inflamed.

A joint attacked by rheumatic fever is swollen, red, hot, tender to touch, and painful. The swelling is most manifest in the knee, where effusion can easily be recognised, in the ankle, in the wrist, and in the joints of the fingers. The colour is mostly a bright pink, and not the dark red of gout and some erythemata; it rarely covers the whole swelling, and may be in patches. The tenderness is sometimes extreme, so that a slight shock on the bed, and any clumsy handling of the joint, will cause intense pain. It may persist after spontaneous pain has subsided. In the shoulder, hip, and elbow joints, pain and tenderness are the chief evidences of rheumatism, as slight swelling is not easily recognised, and redness is generally absent.

It has been stated that the synchondroses are also sometimes involved in rheumatic fever. Undoubtedly the sheaths of the tendons about certain joints are often inflamed, especially those about the wrists and ankles; and some of the redness that extends on to the dorsum of the foot and hand may be due to their inflammation.

With this multiple arthritis there is always associated some *pyrexia*. It is very variable, both in intensity and duration. It does not commonly rise above 103° F., oscillates with some irregularity, and mostly subsides with the inflammation of the joints. It may last nine or ten days if the disease is untreated, or treated inefficiently, but it often ends sooner; and it recurs with any recurrence of the arthritis. It is also influenced by the cardiac lesions, especially pericarditis, or by pleurisy: and it sometimes rises to a great height, becoming thus a dangerous complication (*hyperpyrexia*).

Profuse *sweating* is a characteristic of rheumatism, and occurs without materially reducing the pyrexia. The sweat has a peculiar sour smell, but the reaction is not always strongly acid, and may be even neutral. With this there may be an eruption of the clear vesicles called *sudamina*, or of the vesicles containing a point of pus, and surrounded by a pink areola, known as *miliaria*. The fever is not generally accompanied by much cerebral disturbance, and delirium is not a marked feature in uncomplicated rheumatic fever. The *tongue* is usually large, broad, flabby, and covered with a thick, white, creamy fur. The appetite is bad, and the bowels are constipated. The *urine* is scanty, high coloured, and acid; it contains only occasionally a trace of albumin.

Cardiac Lesions.—In a large proportion—between a third and a half—of the cases of rheumatic fever beginning with joint-inflammation, the heart is afterwards affected: and sometimes the heart is attacked before the joints, or the cardiac lesions may occur without any obvious affection of the joints at all. This greater liability on the part of the heart is especially frequent in children, and as the cardiac lesions are less painful than the articular, there

is a possibility of their being overlooked, which the physician should never forget.

The lesions in question are endocarditis, pericarditis, and acute dilatation, probably from myocarditis. *Endocarditis* commences almost invariably in the valves, and in the valves of the left side. Its occurrence is sometimes marked by increase of fever or by some præcordial distress, or by quickened action of the heart: but in the great majority of cases it is revealed only by auscultation, when a soft bruit may be heard muffling or replacing the first sound of the heart, either at the apex or at the base in the aortic area. A hæmic murmur, systolic in rhythm and rough in quality, is sometimes heard in the second left intercostal space, and must not be mistaken either for aortic endocarditis or for a basal pericarditis; the patient is often, but not always, anæmic. The murmur indicative of endocarditis may disappear in the course of the illness, or may persist into convalescence. Exceptionally, pronounced heart-failure shows itself within a few months of the rheumatic attack. *Pericarditis* may accompany endocarditis, but is very much less frequent; its onset is more often attended with subjective symptoms, such as præcordial pain or distress, local tenderness, rapid action of the heart, and occasionally considerable elevation of the temperature. Friction sound is usually the first physical sign, and increase of præcordial dulness from effusion soon follows: the dulness may extend upwards to the first intercostal space, an inch or more beyond the left nipple, and an inch and a half to the right of the middle line; but the effusion is rarely sufficient to prevent the persistence of the rub as long as the inflammation continues. *Myocarditis* occurs as a part of rheumatic carditis, and as a consequence of the toxins of the disease; and it is no doubt as a result of this that the acute dilatation takes place which is occasionally seen, causing rapid, irregular action of the heart, dyspnœa, vomiting, and delirium. It is often accompanied by pericarditis, and unless quickly subsiding or controlled, is likely to be fatal. It is probable, however, that in a milder form it frequently occurs, and it may be, as some think, that the apical murmurs heard in acute rheumatism are due to dilatation from myocarditis, and not to endocarditis.

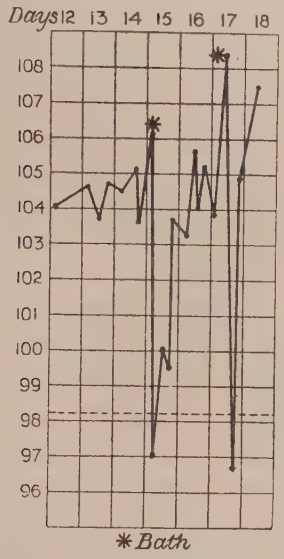
Pleurisy with effusion is often seen in association with pericarditis; it may be single or double, and if single is mostly on the left side. The patient may complain of pain, but the pleurisy is often first noticed by observing the markedly high thoracic breathing of the patient, when an examination of the bases will show, on one or both sides, dulness, deficient tactile vibration, weak vesicular murmur, or soft high-pitched bronchial breathing.

Similar physical signs are occasionally brought about in cases of great pericardial effusion, by compression of the left lung from the front by the distended pericardial sac. Lobar *pneumonia* is quite rare in rheumatic fever; *bronchitis* is more common.

The other lesions deserving of mention are *tonsillitis*, which sometimes occurs early in the case, *pharyngitis*, and various *cutaneous*

lesions. Besides the sudamina and miliaria already mentioned, different forms of erythema may occur, especially *E. marginatum*, and *E. papulatum*. *E. nodosum* is also by some regarded as of rheumatic origin. Occasionally one sees a purpuric eruption complicating rheumatism (*peliosis rheumatica*). This appears mostly on

FIG. 11



Hyperpyrexia in Rheumatic Fever.
Fatal Termination.

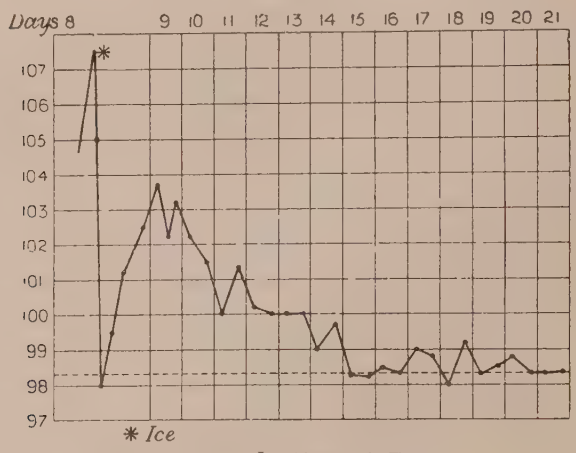
the feet, ankles, and lower parts of the legs, as a more or less continuous bright red eruption, made up of numerous small red petechiæ, which do not disappear under pressure. They commonly last only a few days, and give place to brown or yellow staining as they subside. Sometimes the purpuric spots are much larger and more generally scattered. Another lesion seen in rheumatism, especially in children, and often in association with chorea and cardiac lesions, consists of small *subcutaneous nodules*, which occur in the neighbourhood of joints, and over bony ridges and prominences elsewhere. They are freely movable under the skin, and slightly on the fibrous structures beneath them, and consist of wavy fibrous tissue, with spindle-shaped nucleated cells, and some vessels. They last a variable time, and may disappear in a few weeks.

On the side of the nervous system two important complications may occur, namely, *Chorea* and *Hyperpyrexia*. The relations of the former to rheumatic fever will be more fully discussed later (*see* Chorea); but it certainly sometimes occurs in immediate connection with the

fever and synovitis, commencing before or in the course of the pyrexia, perhaps terminating about the same time, or prolonged for many days or weeks after.

Hyperpyrexia is a very dangerous but happily rare complication, which was formerly described as cerebral rheumatism; the symptoms,

FIG. 12



Hyperpyrexia in Rheumatic Fever.

Recovery.

indeed, point to grave cerebral disturbance, but they are probably the result alone of an excessively high temperature, and lesions of the brain or its membranes are rarely observed. Hyperpyrexia is not restricted to any particular class of case; it may supervene equally in one that appears well on the road to recovery, and in one that has threatening lesions of the chest. It is not determined by any considerations of age, sex, occupation, previous illnesses, climate, or season; but it seems to occur with greater frequency in some years than in others.

In a certain proportion of cases, some warning is given; the joint-pains subside rather suddenly, and the sweating ceases; the patient becomes restless, and, after a few hours, talkative or even delirious. The temperature, formerly perhaps under 100°, is now found to have risen to 105° or 106°, and, unless measures be quickly taken to reduce it, it rapidly reaches 107°, 108°, even 110° and 111°. The delirium is at first moderately active, the muscles twitching, and the eyes restless; the patient may try to get out of bed. The face is generally dusky red, and the tongue dry and tremulous. When the temperature exceeds 107°, the patient becomes semi-comatose, or even quite comatose, a condition which is always present if the thermometer marks 110°. Under suitable treatment for reduction of the temperature, recovery often takes place; but otherwise the

respiration becomes more frequent and shallow, the face more dusky or livid, the pulse rapid and feeble, râles accumulate in the chest, and death ends the scene, often within twelve or twenty-four hours of the first indication of hyperpyrexia. Even if the temperature is reduced to the normal it may rapidly rise again: and this alternation may occur five or six times, either to end in recovery or in death by exhaustion.

Course of Rheumatic Fever.—If untreated, the symptoms may last from ten to fourteen days, when they will often subside; if treated by the method now usual, the pains and fever are often gone within a week. In any case, however, rheumatism shows a great tendency to *relapse*, the joints being affected in a precisely similar manner after an apyretic painless interval of from two days to a fortnight. In such a relapse the patient runs just the same risk in regard to the heart, and to the occurrence of hyperpyrexia, as he does in the first attack. Another relapse may succeed, or irregular affections of now one, now another joint, with or without marked pyrexia. Sometimes recovery is delayed by the persistence of the inflammation in one joint for weeks or months; pain, swelling, and stiffness are prominent troubles, and the joint has ultimately to be dealt with on surgical principles, with rests, splints, and local treatment. Another cause of delay in convalescence has already been mentioned—namely, the rapid progress of an endocarditis, so that the patient passes at once from rheumatism into pronounced heart-disease, with murmurs of aortic or mitral disease, and failing cardiac muscle.

Death takes place in the course of an attack chiefly from hyperpyrexia or from the thoracic complications, especially when endocarditis, pericarditis, and pleuritic effusion on one or other side occur simultaneously.

Morbid Anatomy.—The joints have been found in fatal cases to contain a turbid synovia, with shreds of fibrin. Leucocytes are present, but the fluid is never purulent. The synovial membrane itself is vascular, and covered with a layer of lymph. Probably the joint changes are even slighter than this when such rapid subsidence takes place as is often witnessed. In the tendon-sheaths have been found opaque serum and greenish-yellow lymph. In cases dying with thoracic complications are seen the characteristic lesions described under Endocarditis, Pericarditis, and Pleurisy. Where hyperpyrexia is the cause of death, there are not necessarily any lesions, other than such slight changes as may be recognised in the joints. The lungs are mostly congested, and so are other organs, but beyond this they may be perfectly healthy; there may be no pneumonia, no pleurisy, no endocarditis or pericarditis. When lesions are present, the most common are pericarditis, endocarditis, myocarditis, and dilatation, pleurisy, pneumonia, and softening of the liver, spleen, or kidneys. Meningitis was found in two out of twenty-four cases of hyperpyrexia noted in a report on the subject by the Clinical Society. In some cases of rheumatic fever with purpura, the

intestine has presented an extremely congested and ecchymosed mucous membrane.

Diagnosis.—This usually presents no difficulty, the acute occurrence of joint-pains, with redness and swelling, fever and profuse sweating, being mostly decisive, especially if it occurs in young persons with previously good health, or, on the other hand, with a previous history of rheumatic fever or of heart-disease. But multiple arthritis is the result of many infections, and almost any one of the acute varieties may occasionally be confounded with rheumatism. If multiple synovitis occurs in women after confinement, it may be of a septicæmic or pyæmic nature; and in other circumstances *pyæmia* may give rise to joint-affections deceptively like rheumatic fever. In the latter, the inflammation is often of short duration in each joint; in the former, the joints once attacked only slowly recover. *Gonorrhœal synovitis* is also more persistent than rheumatic fever, and it is only rarely accompanied by cardiac complications; but in early stages may be readily mistaken for it, until the presence of a discharge is ascertained. Some diseases, in which the joints are not mainly involved, may for a time be confounded with acute rheumatism, chiefly on account of pains in the limbs, as well as fever; for instance, *enteric fever* in its early stage, *relapsing fever*, and *acute infective osteomyelitis*. A careful examination of the limbs will generally show that the joints are not the only parts of the limbs involved, as they are in rheumatic fever. The diagnosis from *gout* will be given with the description of that disease.

Prognosis.—The mortality of rheumatic fever is small. Most danger is to be apprehended from the coincidence of endocarditis, pericarditis, and pleural effusions, and from the occurrence of hyperpyrexia. In the former, recovery may take place even with extensive effusions; those into the pleura are less to be feared than those into the pericardium. Hyperpyrexia is dangerous in proportion to the temperature reached before cooling measures are adopted; but repeated rises of temperature after reduction may eventually be fatal. In any case the possibility of future cardiac disease has to be remembered.

Treatment.—For the efficient treatment of even mild cases of rheumatic fever, rest in bed is absolutely necessary: in severe cases the patient cannot do otherwise than lie still. The joints should be protected from every risk of injury. Sometimes it is desirable to raise the bedclothes from the limbs by a cradle; and some local relief to the pain may be obtained by wrapping them round with cotton wool, upon which, in severe cases, a little anodyne, as belladonna or opium liniment, may be sprinkled: or methyl salicylate (artificial oil of wintergreen) may be spread on the joint and covered with guttapercha tissue. The diet should consist mainly of milk: indeed, if it is well borne, it may be given alone, or diluted with barley-water, lime-water, or soda-water.

The drugs now almost universally employed are salicin, salicylic acid, and salicylate of sodium. When the patient is fully under the

influence of one of these drugs, the pains disappear, the redness and swelling of the joints subside, and the temperature falls two or three degrees—it may be to the normal. If the drug is then lessened or discontinued, the pains will most likely return; if the dose is maintained, the rheumatism may be practically cured from that time; but the treatment, both by drug and diet, will have to be continued for ten days or more, at the end of which time some relaxation may be cautiously allowed. An efficient dose of either salicylic acid or its sodium salt is 20 grains, and of salicin 30 grains, every four hours during the first twenty-four or thirty-six hours; but in less severe cases a smaller quantity may suffice. Some give a smaller dose every hour for the first four or five hours, and then diminish the frequency to every two hours. If the attack is very severe it may be desirable to give a 20-grain dose every two or three hours for the first day. If too much is given, the patient suffers from headache, deafness, tinnitis aurium, and slight delirium, which cease when the drug is withdrawn. Occasionally vomiting, a slow or irregular pulse, albuminuria, epistaxis, or hæmaturia has occurred. As a rule, the earlier toxic symptoms coincide with the subsidence of the pains; but this having been obtained, the frequency of the dose must be reduced to four times or three times a day, at which rate it should be continued until five or six days have elapsed from the last pain or the last abnormal temperature, when the drug may be stopped altogether. About this time, also, the diet may be increased by the addition of farinaceous food, and, after a few days, meat-broth, fish, and finally meat, may be given. If, however, there is any return of the rheumatic symptoms, the diet must again be reduced to milk alone for a time.

There is no material difference in the effects upon rheumatism of the three drugs under consideration. The sodium salt is generally preferred, but salicin is said to produce toxic symptoms less readily, and it is less depressant. Sometimes these drugs are not so successful; the pains continue in abated form, or relapses frequently occur. Salicylate of quinine (2 to 6 grains) may then be useful; or recourse may be had to the old alkaline treatment—potassium bicarbonate or acetate, 20 grains every four hours; or to potassium bicarbonate with quinine. There are several other compounds containing salicylic acid, such as salol, salophen, and aspirin, which have some influence upon the pains of acute rheumatism. Aspirin or salicylacetic acid has been largely used, in 10 or 15 grain doses in cachet.

The treatment of the cardiac complications is described under Diseases of the Heart. Prolonged rest in convalescence is desirable after such complications to diminish any strain upon the valves or cardiac muscle. Pleural effusions, even if abundant, generally subside, and as a rule need not be tapped.

The treatment of hyperpyrexia must be prompt and energetic; it consists in the application of cold externally whenever the temperature is found to be rising above 105° F. Salicylic acid and other antipyretics do not lower the temperature with sufficient

rapidity, and the best method of reducing it is by the immersion of the patient in a water-bath at a temperature of 80° or 90°, which may be further cooled to 70° or 60° by masses of ice, if necessary. The patient, even though comatose and apparently moribund, must be placed in the bath and kept there from ten to twenty minutes, or until his temperature has fallen below 100° F. By this time he generally shows signs of returning consciousness. He should then be placed in bed; the extremities may be kept warm, but only light coverings should be placed on the body; a little brandy may be given, and the temperature carefully taken every half-hour. A rise to 104° should again be met by the bath. If at any time a bath is not available, the body may be cooled by packing it in sheets wrung out of ice-cold water, or by rubbing lumps of ice over the surface of the body until the required effect upon the temperature is produced. The fall of the temperature is often accompanied by an increase of the joint-pains, and the rheumatic fever may afterwards continue in the ordinary form.

GONORRHŒAL SYNOVITIS

The acute inflammation of the urethra, known as *gonorrhœa*, is an infective disease, of which the micro-organism is known as the *gonococcus*. Its effects are not always confined to the urethra, but in some cases spread to adjacent parts, causing inguinal bubo, orchitis, and cystitis; and in other cases to more remote parts, causing pleurisy, peritonitis, synovitis, infective endocarditis, suppurative synovitis, or other lesions.

Gonorrhœal synovitis, from its resemblance to acute rheumatism, requires separate notice. It begins at an interval of fourteen days or three or four weeks from the commencement of the urethral discharge, sometimes while the discharge is still purulent, more often during the subsequent stage of gleet.

Symptoms.—In *acute* forms of gonorrhœal synovitis several joints are at first affected with pain and swelling, but the disease soon localises itself in one only, which is most frequently, according to Davies-Colley, the elbow, but may be the knee, ankle, wrist, or foot. There is very extensive redness, with swelling, pain and tenderness. The redness often spreads up the limb far beyond the joint, and the tissues are infiltrated to a corresponding extent. This may be such that it is actually mistaken for abscess, and it may have for the same reason a closer resemblance to gout than to rheumatic fever. The pain is very severe on the slightest movement; the fever is not high. The inflammation only slowly subsides, and leaves a good deal of stiffness behind; but the joint does not often suppurate. Cardiac complications are only occasionally observed. Davies-Colley stated that this form of gonorrhœal arthritis was as common in women as in men.

In other less acute or *subacute* cases the resemblance to a mild rheumatic fever is in some respects closer; the joints are swollen, not

so red, and less generally infiltrated. All the joints in the body may be affected; but the knees, ankles, and wrists are most often involved. Not infrequently there is much pain in the fasciæ, especially in the plantar fascia; and conjunctivitis and sclerotitis occur in a certain proportion of the cases. As in the acuter forms, the inflammation tends to be persistent and does not readily subside and come again, as it does in ordinary rheumatic fever. It lasts two, three, or more weeks, and leaves a great deal of stiffness or even fibrous ankylosis.

Anatomical Changes.—These are serous effusion into the joint, infiltration and œdema of the tissues around it, and in severe cases suppuration, erosion of the cartilages, disorganisation of the joint, and ankylosis. The synovial membrane is primarily affected in the subacute cases, and the surrounding fibrous tissues are chiefly and first involved in the acute forms.

Pathology.—Organisms closely resembling or identical with the *gonococcus* have been found sufficiently often in the fluid of the diseased joints to make it clear that the lesion is a specific result of gonorrhœal infection, and not a part of either rheumatic fever or of pyæmia.

Diagnosis.—The disease is most likely to be mistaken for *rheumatic fever* until it is discovered that the patient has a discharge; or until the persistence of the arthritis in a few joints makes one suspect the nature of the case. The resemblance may be increased by a history of previous attacks; for though gonorrhœal synovitis does not itself recur after long intervals like rheumatic fever, other attacks are often induced by fresh gonorrhœal inflammation. The acuter forms of the disease may closely resemble *erysipelas, abscess,* or *acute gout*. The age of the patient, and the position of the inflammation, will generally exclude the latter. *Pyæmia* may be a cause of multiple synovitis after gonorrhœa; but in this case the illness is generally more severe, with rigors, and such serious complications as pericarditis, endocarditis, pneumonia, or pleurisy.

Treatment.—It is, no doubt, desirable to cure the urethral discharge as soon as possible. For the arthritis alkalies and iodide of potassium have been largely employed, the latter in full doses; but it is probably better to give plenty of good food, with cod-liver oil, and iron or cinchona. Serum and vaccine treatment have also been employed. Vaccines are obtained by cultivating the gonococcus from the patient's discharges, and cocci in gradually increasing doses are injected, checked if possible by the opsonic index. Even then the cure often takes five or six weeks. An antigonococcus serum has also been used with success in a dose of 25 c.c., subcutaneously injected for four or five days. Fenwick and Parkinson have had good results from the injection into the rectum of a polyvalent antistreptococcus serum, using a first dose of 10 or 20 c.c., and subsequent doses of 10 c.c. daily. Locally the joints may be painted with iodine. In acute cases the limb should be kept completely at rest by means of a plaster of Paris splint; and anodyne applications,

especially the compound mercury ointment, with extract of belladonna, may be used. As soon as the inflammation has subsided, the tendency to fixation must be met by friction, shampooing, and passive movements.

TETANUS

In this disease, of which the name is derived from τείνω, I stretch, the essential condition is the occurrence of tonic contractions of most of the muscles of the body, with paroxysms of increased contraction from time to time. It is due to a bacillus, the bristle bacillus of Nicolaier (*B. tetani*), which exists in different forms of earth, or garden mould, and which will cause tetanus in animals when such earth is inoculated under the skin.

Ætiology.—It occurs in quite young infants (*tetanus neonatorum*), and after that age, at all periods of life from five years upwards. It is more common in hot countries, and the dark-skinned races seem especially liable to it. A very frequent antecedent is injury (*traumatic tetanus*), by which an entrance is provided for the bacillus. This may be of any kind, from a mere scratch with a pin or nail to the most serious compound fracture or lacerated wound; but infection is especially liable to take place when the wound has been contaminated by contact with earth, dirt from the road, garden mould, stable straw, or similar materials. In new-born infants the organism enters by the cut surface of the umbilical cord; not infrequently, especially in tropical climates, the disease follows upon abortion or labour at full term. Even after operations under antiseptic conditions tetanus has occurred. Probably, in all cases formerly called *idiopathic*, some means of local infection was overlooked. For instance, a stableman with otorrhœa had tetanus, no doubt infecting the meatus and tympanum with his finger soiled with stable-dirt. The disease is sometimes epidemic.

Symptoms.—Within a few days of the occurrence of the injury in obviously traumatic cases, and without any warning in others, the patient feels stiffness at the back of the neck, and the same in the jaws, so that he is unable to open his mouth wide, or to masticate properly. He may continue like this for a day or two, or may rapidly pass on to the next stage, in which there is rigidity of the muscles of the trunk and to a less extent of those of the extremities. The back becomes rigid, and is slightly arched, with the concavity backwards (*opisthotonus*); the muscles of the trunk and abdomen become quite hard from constant contraction; the movements of the chest are limited from the same cause; the legs are generally rigid, but the arms are only rather stiff about the shoulders and elbows, and the fingers may be moved freely. By this time the jaw is generally firmly fixed by contraction of the masseters, and the teeth cannot be separated for more than a quarter of an inch (*trismus*, or lockjaw, by which last name the disease itself is popularly known); the angles of the mouth are drawn outwards, and the lips

are slightly separated; the eyebrows are drawn up by the frontal muscles, and together by the corrugators, so that the facial expression is that of a painful grin, known as the *risus sardonicus*. When this stage has been reached, the so-called "spasms" or paroxysms of increased and even violent muscular action begin. These consist of sudden contractions involving the whole of the muscles hitherto in tonic rigidity. The teeth are clenched more violently, the *risus* becomes more marked, the head is thrown back, and the back arched more strongly, the chest is fixed, and the respiratory process is checked; a groan may escape from the patient, either elicited by pain or the result of expiratory spasm. The paroxysm is often of momentary duration, scarcely to be counted in seconds, and the patient relapses into his former condition of tonic contraction; or it may last several seconds, with imminent danger to life, from the hindrance of respiration. It is always intensely painful; it is brought on by external impulses, by touching the patient, jerking his bed, by passing a catheter, or giving a subcutaneous injection. The paroxysms occur at first at intervals of half an hour, an hour, or more, but as the disease progresses unfavourably, they become more violent, and occur at shorter and shorter intervals. Between the spasms there is still some pain from tonic contraction, respiration is not entirely free, and the voice is feeble. The reflexes are increased. The pulse is small and quick, and becomes quicker during the paroxysms. The temperature generally at first remains normal, and may continue so to the end, though it sometimes rises a little before death; sometimes the temperature is constantly above normal; in other cases a hyperpyrexia of 107° or 108° occurs just before death, and the temperature has been observed to continue rising even after death to 110°. The urine is often retained, so as to require the use of the catheter. Sensation is generally unaffected, and the cerebral functions are mostly quite normal until near the end, when delirium may occur. In a great number of cases the disease progresses to a fatal termination in from one to twelve days: the paroxysms become more violent and frequent; and death takes place from exhaustion, or from spasm of the glottis, or from fixation of the respiratory muscles; or pneumonia or bronchitis adds its influence against the patient. As happens both in fatal chorea and in hydrophobia, the muscular contractions sometimes entirely cease for eighteen or twenty-four hours before death. In a few cases life is prolonged to the third or fourth week. On the other hand, recovery may take place: the spasms, having reached their height, gradually become less frequent; the constant rigidity of the muscles subsides, and the patient is convalescent in from three to six or eight weeks.

Varieties.—Occasionally a case runs its whole course to a fatal termination without any paroxysms, in addition to the general rigidity. Very rarely paroxysms occur without the continuous spasm. The names *cephalic* tetanus and *hydrophobic* tetanus are given to cases which arise from injuries to the head, involving the

distribution of the fifth nerve. The peculiarities of this variety are that the facial nerve is paralysed, and that spasms of the throat occur resembling those of hydrophobia. There is no essential difference between *acute* and *chronic* tetanus.

Morbid Anatomy.—Many cases present after death no pathological lesions whatever. The organs most commonly affected are the lungs, which may be the seat of pneumonia, bronchitis, œdema, or hæmorrhages. The central nervous system, as a rule, looks perfectly normal to the naked eye. Microscopical examination also often fails to reveal anything, but sometimes shows minute changes, which, however, do not in any way explain the clinical conditions, and are partly, no doubt, the result of the disturbance of the circulation during the paroxysms. The muscles of the trunk, especially the abdominal muscles, are sometimes ruptured, or the seat of hæmorrhages. In traumatic cases the state of the wound bears no relation to the final result—it may be healing, or healed, or suppurating, or sloughing.

Pathology.—The bacillus multiplies chiefly in the neighbourhood of the wound, and produces poisons which have an affinity for the central nervous system, especially the spinal cord; and these, circulating in the blood, or conveyed along the nerve-trunks, act directly upon the nerve-cells. The bacilli have, however, been found in the inguinal glands. Tetanus has also been transmitted from man to animals by the inoculation of materials from the wound, and by the injection of urine which contains the toxin.

Diagnosis.—Tetanus may have to be distinguished from strychnia-poisoning, hydrophobia, spinal meningitis, tetany, muscular rheumatism, and hysteria. In *strychnia-poisoning* the extremities are involved to much greater extent than in tetanus, and the paroxysms are excited by external stimuli; but in the intervals the muscles are relaxed. The symptoms develop very rapidly, but do not begin with trismus. In *hydrophobia* there is no continuous rigidity; the spasms involve the respiratory muscles, and are excited by the attempt to drink, or the sight of fluids. Mental agitation or even maniacal excitement is generally present. In *spinal meningitis*, again, trismus is not an early symptom, nor is there constant rigidity; muscular spasms are excited by attempts to move, and the temperature is high from the first. The early occurrence of cerebral symptoms would be opposed to tetanus. The peculiar distribution of the spasm in *tetany* makes it easy to distinguish it from tetanus. *Muscular rheumatism* may cause stiffness of the back of the neck, which might, under certain circumstances, cause alarm; but trismus is never present. In severe forms of *hysteria* opisthotonus is often a prominent feature, but it occurs as part of a series of convulsive movements, which cannot be mistaken. Trismus also may occur in hysterical patients, but is not accompanied by rigidity of the cervical muscles, and is variable in intensity. Other indications of hysteria, as, for instance, a preceding convulsive fit, may be present.

Prognosis.—Amongst traumatic cases about 90 per cent. die, and of other cases about 50 per cent. Tetanus is very fatal after pregnancy and abortion. On the whole, recovery is less common in cases due to severe and extensive injuries than in those due to slight injuries; and it is less frequent when the symptoms develop rapidly after the injury, or run a rapid course to a severe stage, than when they come on in every way slowly.

Treatment.—The patient should be kept at rest, and is best placed in a darkened and perfectly quiet room, so as to avoid all impressions of sight and sound. Nourishment should be given freely, in fluid form; but the closure of the jaws may necessitate its being given by a nasal tube, or by the rectum. If there is already a deficiency in the teeth, a tube may be passed into the mouth; but the extraction of a tooth for this purpose is of doubtful expediency. It has been suggested that once or twice daily chloroform may be administered, so as to relax the jaws and enable food to be given. In traumatic cases the wound should be treated thoroughly with antiseptics, so as to remove or destroy as far as possible the bacilli from which the poisonous materials are proceeding. But this is not in itself sufficient, as the appearance of the symptoms is proof that the poison is already circulating.

The treatment of tetanus by an *antitoxin* was attempted by Tizzoni and Cattani long before the use of an antitoxic serum in diphtheria. The preparation of the tetanus serum is very similar, and 10 c.c. should be injected under the skin, two, three, or more times daily. This proceeding has been sometimes successful; but the cases which have recovered under it have been chiefly those, the less acute cases, which would have got well on other plans of treatment. Having regard to the prompt action of the toxins upon the nerve-cells, the antitoxin has been injected into the cerebral tissue through a hole drilled in the skull. This also has given good results, but there is some risk of septic inflammation.

The anodynes and spinal sedatives which have been employed in the treatment of tetanus can only be regarded as palliative. They are practically powerless against the most acute and severe forms; in less violent cases some of them are of benefit by relieving pain or diminishing spasm. But this last effect will only be gained by the employment of full doses, which, in the case of some drugs, must be carefully watched to prevent the production of other toxic effects. Chloral, bromide of potassium, and Calabar bean, have been most successful. Chloral may be given in doses of 30 or 40 or even 60 grains every four or six hours; bromide of potassium in drachm doses frequently; extract of Calabar bean in 1-grain doses every two or three hours; or the sulphate of eserine in doses of $\frac{1}{6}$ to $\frac{1}{4}$ grain every three hours by subcutaneous injection, until its toxic effect is shown in fibrillary twitching of the muscles and diarrhœa. Morphia injections or opium may also be given once or twice daily to procure sleep; but the continuous use of opium is not so successful as that of the above-mentioned drugs. Belladonna

and atropine, aconite, cannabis indica, nitrite of amyl, and curare have also been employed, but with even less encouraging results. In most cases it will be found expedient to give some stimulant with the food.

A recent method of lowering spinal excitability is by the injection of magnesium sulphate solution into the spinal theca: and this is based on the fact that such a solution applied to nerve-trunks abolishes motion and sensation in the parts concerned. For adults 5 c.c. of a 25 per cent. solution of magnesium sulphate are injected by lumbar puncture after removal of a little cerebro-spinal fluid. This produces flaccid paralysis of the legs, and care has to be taken that the fluid does not reach the upper part of the spinal canal, lest respiratory paralysis and death should ensue. The treatment is symptomatic, can only be partial, and is not free from danger.

Prevention.—The prompt cleaning of all soiled wounds is an obvious means. French surgeons have injected tetanus antitoxin in cases of injury immediately on admission to hospital, but this has by no means always prevented the occurrence of tetanus afterwards. It has also been recommended to apply tetanus antitoxin immediately to a wound likely to have become infected with the tetanus bacillus.

HYDROPHOBIA

(*Rabies, Lyssa*)

This is an infectious disease, which is invariably caught from animals. The disease occurs in the wolf, fox, cat, cow, and horse, but much more frequently in dogs; and it is the bite of a dog which, as a rule, by means of the saliva, introduces the poison into the human blood. Rabies in animals occurs in two forms. In the first, or *furious rabies*, the dog is at first low-spirited, timorous, and unwilling to move; he then becomes suspicious and irritable, with a strong tendency to bite, and often with a peculiar howl. He refuses his ordinary food, and will eat straw, earth, hair, clothes, bits of wood, &c. Paralysis supervenes, the lower jaw drooping, the limbs failing, so that the animal can no longer stand, and finally death takes place. In *dumb rabies* there is no maniacal stage; the paralytic symptoms appear early, and are soon fatal. In neither case is there the fear of water which gives the name to the human complaint.

Ætiology.—In only about half the cases of bite by mad dogs does hydrophobia afterwards develop, and it is more likely to be the case if the bite is on an exposed part, such as the face or hand. A portion of clothes driven in by the tooth may protect from infection. If an abraded surface, or even perhaps a mucous membrane, is licked by a mad dog, infection may occur. The disease is more frequent in men than women, from the more frequent association of the former with dogs; it may occur at all ages.

Symptoms.—After the inoculation there is a period of *incubation* which is of remarkable length : in the majority of cases it is from two to nine weeks, and in some cases it is several months. During this time there may be absolutely no symptom. The first definite sign is often an uneasy sensation of pain in the scar of the wound. This pain may be very severe, and the scar may be slightly reddened or tender. But these indications may be entirely absent, and then the first sign is a feeling of malaise or depression, restlessness, sleeplessness, irritability, failing appetite, with a sense of choking or an uneasy feeling about the throat. Then appear the spasms, which are so characteristic of the disease : they are excited by the attempt to drink, by the sight of water or the vessel containing it, or by the suggestion of those around that some fluid should be taken. Later on they are induced by almost any external impression—a breath of air, a flash of light, or a loud noise. The spasms involve the muscles of deglutition, but the most obvious are those affecting the muscles of respiration—a sudden deep inspiration, like a sob or sigh, is made, the shoulders are raised, the chest expanded, and the sterno-mastoids or platysmas contracted. If water is forced upon the patient, more voluntary efforts to reject it are made, and an aspect of fright or terror is assumed. After a time the convulsions extend to other muscles of the body, presenting a tetanoid character. The difficulty of deglutition is shown in another way, for the saliva is not swallowed, but is constantly being collected in white frothy pellets, and is expectorated in all directions. With the increasing severity of these spasms, the patient becomes excitable, talkative, delirious, or wildly maniacal, with delusions and hallucinations. The temperature is raised, the face is flushed, all attempts to give food may be futile—at most a small quantity of milk or other nutriment may be gulped down in a moment of greater control. Emaciation is remarkably rapid in the small time the disease lasts, and exhaustion necessarily follows. Not infrequently, towards the end, the spasms cease entirely, and the patient may even take good quantities of food ; but even if this is so, it does not avail to prevent the fatal end, which may be preceded by paralysis and coma. In a few cases, the final symptoms are paralytic ; motion, sensation, and reflexes are lost in the legs, arms, and other parts.

The disease lasts from two to four days ; a period of ten days seems to be the longest known. Death is almost inevitable in the developed disease.

Anatomy.—There are but few macroscopic changes in the organs ; the cerebro-spinal fluid is often increased in quantity. The microscopic changes found in the nervous system, especially in the cortex of the brain, in the spinal cord, and most abundantly in the medulla oblongata, consist of dilatation of vessels, collections of small cells round the vessels and in the tissues, clots in the vessels, and small hæmorrhages. Leucocytal infiltration has been also seen in the salivary glands and in the kidneys. By inoculation experi-

ments, the virus is shown to be distributed throughout the central nervous system, and in the secretion of certain glands (salivary, lachrymal, mammary, and pancreas). It has also been shown that the virus after inoculation spreads up the peripheral nerves to the central nervous system. The microbe of hydrophobia has not yet been identified.

Diagnosis.—This is not generally difficult, especially if the fact of infection is well authenticated. There is but little real resemblance to tetanus, in which the permanent rigidity of muscle and the absence of mental disturbance are distinctive. Hysteroid conditions may simulate hydrophobia, and may occur where the mind has been much directed to the possibility of hydrophobia coming on. Globus hystericus may be regarded by the patient as a "spasm" of the throat. *Lyssophobia* is a name given to a condition in which hydrophobia is only simulated.

The demonstration of the nature of the disease has been obtained after death by the inoculation into rabbits of an emulsion of the medulla oblongata. The presence of the disease in any animal inflicting a bite can be shown in the same way.

Treatment.—No remedy is known with certainty to have any influence upon the disease when once it is developed. Temporary relief may be given by morphia injections or chloroform inhalations.

Prevention.—On the occurrence of a bite, a ligature should be at once placed above the bitten part (if it is a limb), and the wound may be sucked so long as the mucous membrane is unbroken, and the mouth is frequently rinsed after ejecting the blood. The wound should then, as soon as possible, be cauterised with nitric acid or the actual cautery, or it should be excised; and the patient should be sent for further treatment to a Pasteur institute.

Pasteur's Treatment consists in the inoculation of the virus of dog's rabies, modified by transmission through rabbits, and by subsequent exposure to air. A rabbit is trephined and inoculated under the dura mater from the spinal cord of a rabid dog; the rabbit becomes rabid after fifteen days' incubation. A second rabbit is inoculated from the first, a third from the second, and so on until the period of incubation, which grows shorter with successive inoculations, is reduced to the minimum of seven days. The spinal cords of these rabbits contain the virus in every part; but if a fragment be separated, and suspended in dry air, the virulence gradually diminishes, and disappears in a period of time which varies with the size of the fragment and the temperature of the air. For the purposes of preventive inoculation a number of fragments of the virulent spinal cords of rabbits are kept in separate bottles of dry air, the date of their introduction being noted. If a healthy dog is injected subcutaneously with a certain quantity of one of these cords that have been dried sufficiently long to destroy the virus—say fifteen days; on the next day with spinal cord that has been dried fourteen days; on the next day with spinal cord of thirteen days' drying; and so on, on successive days, until spinal cord is

injected which has only been dried one day; the dog is then found to be incapable of contracting rabies. Similar inoculations are performed upon human subjects, bitten by rabid dogs, as soon as possible after the injury, in order to neutralise the virus and prevent the development of the fatal symptoms. Some modifications have been made in the original process; in the *intensified* method, the inoculations follow upon one another with much greater rapidity.

Antirabic sera have been prepared, but they appear to be only antimicrobic and not antitoxic. They cannot at present replace Pasteur's treatment, but may possibly be of use while this treatment is being developed.

GLANDERS

(*Equinia, Malleus, Farcy*)

Glanders is a disease which affects chiefly horses, mules, and asses, though sometimes other domestic animals, and is occasionally transmitted accidentally to men. Grooms, stablemen, and others in charge of horses are most liable to contract the disease, which, in its acute forms, is a febrile disorder, characterised by special lesions of the nasal and respiratory mucous membranes, by the formation of subcutaneous nodes and the implication of the lymphatic vessels and glands, and by a cutaneous eruption. It also occurs in a chronic form. The term *Farcy* was given to cases in which the subcutaneous nodules (farcy-buds) with the lymphatic lesion were the prominent features; but it is not desirable to have two names for one disease, and glanders is now the appellation generally adopted.

The disease is mostly transmitted to man by accidental inoculation of wounds, cuts, or abrasions, either in grooming a glandered animal or in skinning one dead of the disease; or a horse may bite its groom, and convey the disease by means of its saliva, or may sneeze and discharge some nasal mucus into the eye, nose, or mouth of any one standing near. It is stated that it may be conveyed by eating the raw flesh of a glandered animal, and that it has been caught in this way in menageries. It may also be communicated from man to man.

The bacillus of glanders (*B. mallei*) is found in the nodules; it is about the size of the tubercle bacillus, but is thicker and differs from it in its staining properties. It has been artificially cultivated, and inoculations have been made, leading to lymphatic inflammation and general infection, with the formation of nodules and ulcers on the nasal septum, and nodules in the lungs, in which, again, the bacillus has been found.

Acute Glanders.—The disease begins with malaise, headache, lassitude, loss of appetite, and pains in the joints and limbs. For a time there is often a resemblance to rheumatic fever or enteric fever, or there may be pain in the side or dyspnœa. If a wound or scratch has been infected directly, it becomes inflamed, tense, and painful; and the skin around has the appearance of erysipelas. The sore ulcerates, and discharges a sanious fluid, and the lymphatics in the neighbour-

hood may become enlarged. The more characteristic features of the disease may not appear for a week or more after its commencement, though sometimes earlier. The *eruption* consists of small red papules, upon which vesicles appear; these soon form bullæ, or pustules, of different sizes, up to half or three-quarters of an inch in diameter, hemispherical, flat or depressed in the centre, with serous, purulent, or blood-stained contents. The base of the pustule is inflamed, and infiltrated for some distance round. After a time, the discharge escapes, and an ulcer covered with scab or slough remains. The *nodes* which form under the skin are at first hard and painful, and generally suppurate; they also frequently occur in the muscles. The lymphatic glands are not always inflamed. The implication of the mucous membranes is shown by a discharge from the nose, which is at first a thin mucus, but afterwards becomes thick, viscid, purulent, fœtid, and often blood-stained. It is connected with the formation of tubercle-like nodules on the nasal mucous membrane, which caseate, ulcerate, and may perforate the septum nasi, or destroy the turbinate bones. Other mucous membranes may be affected—*e.g.*, the conjunctiva, and those of the frontal sinuses, the pharynx, the larynx, and the bronchi. In the lung are found deposits which caseate and suppurate, as well as patches of lobular hepatisation.

The progress of the case is generally downwards, with symptoms of a pyæmic or adynamic character. The temperature is high, but may oscillate; the pulse is quick, and the tongue dry and brown. Albumin appears in the urine, low delirium with tremor is succeeded by coma, the breathing becomes more rapid, and death finally ensues, generally in two or three weeks from the commencement.

Chronic Glanders.—Here the local lesions predominate. They consist of ulcers with thick and hard edges, or abscesses about the joints, or inflammatory swelling beneath the skin or in the muscles. A pustular eruption may also occur, but it develops more slowly than in the acute form. The nasal mucous membrane may also be involved, and in some cases emaciation occurs, with hoarseness and pulmonary symptoms, such as cough and hæmoptysis. The average duration of the chronic cases is stated to be four months.

Pathology.—On *post-mortem* examination of acute glanders changes characteristic of pyæmia are often found: increased fluidity of the blood, and abscesses of the lungs, the pyæmia being secondary to the local lesions.

The characteristic lesions of glanders are found in the mucous membranes, the skin, and the lungs. In the nasal mucous membrane, subepithelial nodules occur, from the size of a millet seed to that of a pea, consisting of lymphoid corpuscles, or pus-corpuscles. In a later stage these nodules have suppurated, and left ulcers with yellowish bases. Around these, fresh nodules of infiltration have formed, which go through the same process. If recovery takes place, irregular puckered scars are left. In the lungs, similar nodes form, the centres of which break down into a caseous detritus. These

are accompanied by patches of broncho-pneumonia, which may form abscesses. Similar nodes form in the intestinal mucous membrane, in the skin and subcutaneous tissue, and in the muscles.

Diagnosis.—In early stages the disease may be mistaken for rheumatism or typhoid fever, and later for pyæmia. In chronic cases, syphilis, scrofula, and phthisis may be simulated. In veterinary surgery, the diagnosis is made by the injection into suspected animals of *Mallein*, which consists of the chemical substances present in the artificial cultures of the glanders bacilli. If the animal is diseased, a definite "reaction" with rise of temperature occurs, similar to that produced in man by Koch's *tuberculin* (*see* Diagnosis of Phthisis).

Prognosis is very unfavourable. Only a few recoveries from acute glanders are recorded; and only about half of the chronic cases get well.

The **Treatment** must be supporting and stimulating. Quinine should be given internally; the nasal lesion should be treated with antiseptic injections, such as creosote, carbolic acid, iodine or potassium permanganate lotion. Abscess of the skin should be opened when ready. For chronic cases, carbolic acid, potassium iodide, arsenic, strychnia, and sodium benzoate have been recommended.

ANTHRAX

This term, formerly the Latin equivalent of *carbuncle*, is now generally used to designate a disease which affects various animals, and is communicated from them to man. In animals it is known as *splenic fever;* in man it includes *charbon* of the French, and *malignant pustule* of English writers. Its distinguishing feature is the presence of a bacillus (*B. anthracis*), which can be found in the local lesions, the blood, viscera, and secretions. This is a motionless, short, homogeneous rod, straight or slightly curved, varying from 5 μ to 20 μ in length—that is, considerably longer than the diameter of a blood-corpuscle. The rods multiply by elongating and dividing, and also produce within themselves spores, which subsequently become free and reproduce the rods; but sporulation only takes place outside the animal body. The spores have great vitality, and resist considerable changes of temperature.

Among animals this disease can be conveyed by direct inoculation, probably the bites or stings of insects, or the bites of dogs that have eaten the flesh of animals dying of the disease. It is also transmitted indirectly by animals feeding in damp meadows or on moist soils, where the specific micro-organism contained in the dejecta of previously diseased animals may have been preserved in an active condition. Pasteur thought that the spores of bacilli multiplying around buried carcases might be carried to the surface by earthworms; but this was not confirmed by experiments instituted by Koch.

Infection in man occurs from the living animal, as in drovers, shepherds, and farmers; or from the carcase, and this is much more common. Thus, slaughterers, butchers, and those who have to do with the hides may be infected through a scratch or wound, and, rarely, it may be contracted in eating the flesh of diseased animals. Most frequently, however, in England it occurs amongst tanners and those who have to handle the skins and hides that come from abroad, and among those who deal with wool and hair from the same animals. Thus, wool-sorters, furriers, tanners, and others in like occupations may contract the disease either by direct inoculation through the broken skin, or by inhalation of dust or wool particles proceeding from the goods. Rarely it is transmitted from man to man by direct contact. Rag-sorters engaged in paper manufactories are subject to a disease which is probably internal (pulmonary) anthrax; and the bacillus of anthrax has been found in the viscera. But some observers have attributed this disease to a *B. proteus*, and others to the bacillus of malignant œdema.

Symptoms.—The different forms of the disease are local or external anthrax—*malignant pustule* proper; and internal anthrax, which includes a pulmonary and a gastro-intestinal form. Either of the last two may be combined with the local variety.

Malignant Pustule.—Infection generally occurs through a scratch or abrasion on the face, neck, hands, or arms. After an *incubation* of a few days, or it may be only some hours, the spot itches or burns, and a small pimple appears, which vesicates, and the vesicle bursts and discharges a thin fluid. The base of the vesicles then forms a brown or black eschar, and the skin around becomes red, swollen, and indurated, forming a prominence from one and a half to two inches or more in diameter. Around the central eschar there is often a ring of small vesicles containing serum, and the skin for some distance round may be œdematous, and the nearest lymphatic glands enlarged and tender. For three, four, or five days the patient may feel in his usual health and continue at work; he then becomes feverish, with prostration, delirium, sweating, or diarrhœa, and finally, in many cases, death occurs, preceded by collapse.

In *malignant anthrax œdema* no definite pustule forms, but an œdematous swelling, usually affecting the eyelids. It is otherwise like malignant pustule, and is mostly soon fatal.

Internal anthrax varies in different cases. The early symptoms are generally restlessness, a sense of depression and exhaustion, and vague sensations in the limbs; then acute fever suddenly sets in with the usual symptoms, and, in addition, great prostration, embarrassed respiration, and rapid collapse. To these may be added the special features of the pulmonary or intestinal form.

In the *pulmonary* form, difficult and laboured breathing, with a sense of constriction, cyanosis, and great prostration, are the main features, without much cough or physical signs other than a few rhonchi and râles. The expectoration, if there is any,

may be bloody. Delirium and coma may precede death, or the mind may be clear to the last. This is the *wool-sorters' disease* observed at Bradford and elsewhere.

In the *gastro-intestinal* form there are vomiting, abdominal pain, and diarrhœa, often with blood in the fæces; sometimes dysphagia, and bleeding from the pharynx and mouth. Fever is slight, but dyspnœa and lividity, restlessness and convulsions of epileptic or tetanic character precede the invariably fatal end.

Anatomical Changes.—In all fatal cases there may be found the changes indicative of acute septic disease: ecchymoses in the submucous and subserous tissues, in the substance of the heart, or in other muscles: hæmorrhage or œdema of the lungs, congestion and softening of the liver and kidneys. The spleen is not always enlarged. When the special *pulmonary* symptoms have been present, there are congestion of the mucous membrane of the trachea and bronchi, hæmorrhages into the lungs or under the pleura, swelling of the cervical and bronchial glands with hæmorrhage into or around them, fluid in the pleural cavities, and ecchymosis and gelatinous exudation in the neck and mediastinum surrounding the trachea and mediastinal glands.

In the *intestinal* form the peritoneum contains serum, which is often blood-stained; there is semi-gelatinous infiltration of the mesentery and retroperitoneal connective-tissue; congestion and swelling of the mucous membrane and submucous tissues of the stomach and intestines, in patches of a quarter of an inch to one or two inches in diameter, which are pink and fleshy on section, but on the surface discoloured, or excoriated, or covered with an adherent layer of blood. There are also submucous and subserous hæmorrhages; and the spleen and the mesenteric and lumbar glands are often enlarged.

Diagnosis.—Much depends at first on the knowledge of the possibility of infection, especially in the internal forms. With a well-developed malignant pustule, the central eschar and the surrounding ring of vesicles on a red infiltrated base are characteristic. Bacilli may be detected in the fluid from the pustule, or in the blood, expectoration, or urine. But they are not generally to be found in the blood for some days, though exceedingly numerous in the local sore by the second or third day. The diagnosis may be confirmed by inoculation of a rabbit, guinea-pig, or mouse with the secretions or with blood. The animal dies within two or three days with dyspnœa, dilated pupils, and, perhaps, convulsions; and the blood contains the characteristic bacilli.

Prognosis.—This is very unfavourable in cases left without treatment.

Treatment.—In malignant pustule the most certain cure is to excise the infiltrated part completely, and apply caustic, such as zinc chloride, to the exposed surface. The patient often improves at once, and is soon well.

The injection of carbolic acid into the tumour is sometimes

very successful. A syringeful (20 or 30 minims) of a 2 per cent. solution of carbolic acid in water is injected into each of four points surrounding the central eschar; and the injections are repeated two or three times a day for four or five days. Energetic local treatment may still be successful, even when there is evidence of general infection having begun. Internal anthrax should be treated with quinine and carbolic acid, stimulants, and suitable nourishing food.

Ipecacuanha has been used with some success, both internally in doses of 5 or 10 grains every four hours, and locally to the wound after excision (Muskett, Davies-Colley). For some years in Italy, and more recently in England, Sclavo's serum, which is obtained from the ass after a long period of immunisation, has been used with success in daily doses of from 20 to 40 c.c. injected subcutaneously.

FOOT-AND-MOUTH DISEASE

(*Aphtha epizootica*)

This disease of cattle and sheep is occasionally transmitted to man. The typical feature of the disease in cattle is the formation of vesicles and bullæ on the mucous membrane of the mouth, lips, and tongue. The affected parts become swollen, and the saliva dribbles away. The vesicles break, leaving a gray layer covering the base. Vesicles also appear on the feet round the borders of the hoofs, and they become pustular and produce crusts. In cows, vesicles form also on the udders and teats. There is a moderate degree of pyrexia. The disease lasts about a fortnight, and generally ends in recovery; except in calves, of which 50 to 75 per cent. die. It is thought that this is due to the milk drawn from the diseased cow irritating the bowel as well as conveying the virus.

The disease appears to be conveyed to man by direct inoculation, and by drinking milk from an infected cow.

The *incubation* is from three to five days. Slight pyrexia and loss of appetite first occur, then vesicles are observed in the mouth, on the lips, tongue, fauces, and hard palate. They reach the size of peas, become opaque, break, and form shallow ulcers, with a dark red base. The lips become swollen, and saliva and mucus are more abundant than normal. Mastication, swallowing, and talking are somewhat painful. There may be some diarrhœa and abdominal pain.

Sometimes vesicles form on the fingers, especially about the nails; they become pustular, and run together; and similar vesicles have been described as occurring on the toes, and on the nipples of women. The duration is from ten days to a fortnight, and the disease is rarely fatal.

Treatment.—Solutions of borax may be used to the mouth, and

painful ulcers should be touched with solid silver nitrate. Zinc or lead ointments or lotions should be applied to the eruptions on the fingers and toes.

ACTINOMYCOSIS

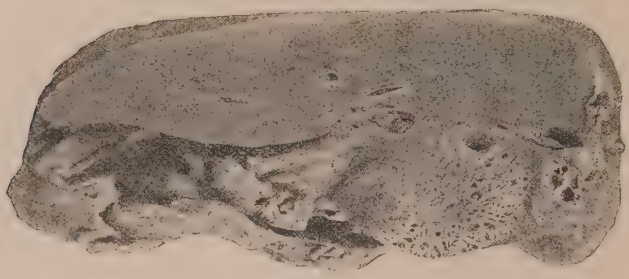
Actinomycosis is due to the entrance into the body of a vegetable parasite, the *Actinomyces* or Ray-fungus. In 1877 Israel of Berlin described the first cases in man, and in 1878 Ponfick showed the identity of the human cases with a similar affection occurring in cattle.

The actinomyces is an organism belonging to the *Streptothrix* group (*see* pp. 11, 21), and is now known as *Streptothrix bovis communis*. It forms masses which are visible to the naked eye as yellow, greenish yellow, or gray, glistening, spherical, granular bodies, mostly about one-fortieth of an inch in diameter, but sometimes as much as one-twelfth of an inch, and consisting, under the microscope, of a central mass of closely woven mycelial threads and cocci, from which proceed radially, in every direction, multitudes of threads, some of which divide dichotomously, finally terminating in club-shaped extremities. Although it occurs in domestic animals, there is no evidence that it has ever been conveyed from man to man. Animals and men undoubtedly obtain it from some common source, such as vegetable food; Boström has actually found in the centre of some of the lesions in man fragments of barley or other cereal, showing almost conclusively that the eating of raw grain has been the means of the introduction of the fungus into the body. Once introduced, the organism adheres to some point of the surface of the alimentary or respiratory passages, penetrates then to deeper parts, and forms local lesions in different parts of the body. These consist mainly of inflammatory changes, of more or less intensity, set up around the granules, so as to form slowly growing tumours, which ultimately suppurate, break down, and discharge. Section in early stages shows:—in the centre the radiating structure of the organism; immediately around it a thick layer of leucocytes, in and among which some of the club-shaped ends of the threads are embedded; and a layer of fibroid connective tissue, forming the periphery of the tumour. From the continued growth and multiplication of the parasite at one spot—as, for instance, in the liver—large tumours may be formed, three or more inches in diameter, consisting of a kind of cavernous tissue, the trabeculæ of which are fibroid, while the spaces either correspond to the organism and leucocytes, as above described, or contain pus, in which the yellow granular masses of fungus lie loose. A remarkable feature of the disease is the way in which lesions extend by contiguity from tissue to tissue over long periods of time; but occasionally the parasite is conveyed by the vessels to remote parts, and a more widespread deposit by *metastasis* occurs.

Symptoms and Course.—These depend on the seat of the primary invasion.

In many cases this is the *mouth*, when a tumour is generally first noticed under the skin over the lower jaw, or on the edge of the jaw. It is hard, does not affect the skin, is chronic in its course,

FIG. 13



Sagittal section of a liver showing the lobulus Spigelii honeycombed by Actinomycosis.

varies in size from time to time, and tends to migrate gradually from the edge of the jaw down to the neck, leaving, for a time, a narrow band of firm tissue in its track. The tumour may shrink up in part, the inflammatory tissue cicatrising; but newer portions continue to form, and ultimately the skin becomes involved, obscure fluctuation is felt, and a thin, sero-purulent, odourless fluid, containing the characteristic granules, is discharged. A sinus is formed, which rarely closes, but continues patent with slight secretion. There is evidence to show that in these cases the parasite has entered by a carious tooth, and this is held to explain the occurrence of the tumours in connection more frequently with the lower than the upper jaw, and with the back part rather than the front part of the lower jaw. The tumour may form in the substance of the lower jaw, and expand the bone. Invasion by the upper jaw results in tumours of the cheek or temple; and an extension to the base of the skull or the mediastinum by means of the pharynx is a possibility which makes the implication of the upper jaw more serious than that of the lower. Penetration of the *œsophagus* has led to mediastinal abscesses, and erosion of the vertebræ.

In *intestinal* actinomycosis, the mucous membrane presents on its surface patches of whitish material, covered with yellow and brown granules. The patches are about two-fifths of an inch in diameter and one-fifth of an inch thick, and adhere firmly to the membrane. The disease may also cause swellings in the substance of the intestinal wall, from which it may perforate into the peritoneal cavity, or, by means of adhesions, invade adjacent viscera or the abdominal wall at almost any point. The *liver* is often secondarily infected in intestinal cases, and then contains large prominent

masses, having the structure above described. Clinically, such tumours may present the characteristics of hepatic abscesses, with local pain, tenderness, remitting fever, and rigors.

When actinomycosis affects the *lungs* the symptoms may be bronchitic or pneumonic. In the former case there may be a close resemblance to putrid bronchitis, the sputum separating into two layers (not into three, as in fœtid bronchitis), the upper clear, and the lower turbid; the latter containing the ray-fungus. When the substance of the lung is affected, pneumonia occurs in patches, the patients cough and lose flesh, and the expectoration is either thick and muco-purulent, containing the typical granules, or it may be viscid, translucent, and rusty, like pneumonic sputa. There is often a certain resemblance to phthisis, but the posterior and lateral portions of the lungs are involved, not the apices; and the sputum is, of course, free from tubercle bacilli. If the inflammatory lesions reach the surface they set up pleurisy or pericarditis. Effusion takes place, or the lung becomes adherent to the chest wall, which then becomes involved, and ultimately soft diffused inflammatory swellings appear on the chest, which may fluctuate, break, and discharge purulent fluid containing the fungus. From the lung, also, the inflammatory track of the organism may stretch through the diaphragm into the abdomen, or behind the diaphragm to the psoas and iliacus muscles, or between the ribs to the surface of the chest. In a case of this kind recorded by Pringle, there were large, soft, fleshy, sarcoma-like growths on the back of the chest, of mottled, purplish-red and yellow colour, covered by very thin skin, and presenting small ulcerative openings, from which a sticky fluid oozed, and in which lay a purulent fluid containing actinomyces granules. These processes are commonly very slow, and are accompanied with varying amounts of fever in different cases.

A primary infection of the *skin* through direct injury by straw, or husks, is much more rare. The lesions are infiltrations resembling those of syphilis or tubercle: sometimes extending deeply into the subcutaneous and muscular tissues.

Infection by the *female genital tract* with extension to the ovaries and Fallopian tubes is also recorded.

Diagnosis.—This can only be made with certainty by the detection of the characteristic granules in the secretions, whether pus, sputum, or urine. Some care is required in looking for them. Pus may be shaken up with a little salt and water in a test-tube, when the granules will come into view. One of these may be then placed on a slide and covered with thin glass, when a low power will show the characteristic radiating structure. Staining reagents may be employed, as, for instance, picro carmine; or the filaments may be stained by gentian-violet or carbol-thionin; and the clubs later by solution of rubin or picric acid. They are stained also by Gram's solution. Actinomycosis of the brain has been detected by lumbar puncture, the fungus being recognised with the microscope in the sediment from the fluid.

The inflammatory lesions accessible to palpation are said to present a special "wooden" resistance.

Treatment.—*Potassium iodide* has a powerful influence in the treatment of actinomycosis, and very remarkable results have been obtained under its use both in bovine and human cases. It should be given to the extent of two, three, or four drachms daily; and better at short intervals of two or three hours during the twenty-four hours, than in two or three doses or at longer intervals (*see* p. 145).

Some cases may require to be treated surgically, and cures have been effected by complete extirpation of the growth, or by scraping out the resulting abscesses and sinuses, so as to remove completely all fungus granules. Visceral lesions are, of course, less suited for this treatment. But in any case the potassium iodide should be pushed.

DISEASES OF THE NERVOUS SYSTEM

Under this heading we have to deal with disorders of the brain, spinal cord, and nerves—disorders which manifest themselves through the functions of motion, sensation, the special senses, and the intellect and emotions.

The nervous system is liable to similar lesions with the rest of the body. It has been already shown to suffer from the toxins of infectious diseases, either in common with the rest of the body, as in typhus or enteric fevers; or more apart, as in tetanus, hydrophobia, beri-beri, and leprosy. Still more localised inflammations and degenerations may take place in the nervous system, tumours may grow in various situations, or the different parts may be crushed or injured. And it is a consequence of the specialisation of almost every nerve or nerve-centre for a particular function, that when damage is done by injury, inflammation, or tumour, the symptoms it produces depend very much, or entirely, upon the precise *locality* in which it occurs. The symptoms associated with pneumonia are very much the same whatever part of the lung is involved; but the effect of a limited lesion in the nervous system may be for a long time harmless in one spot, and rapidly fatal in another. The lung subserves mainly one function; the brain and spinal cord a great number. It is the *localisation of functions* in different parts of the nervous system which enables us to determine the position of the *disease* when it occurs; and as for this purpose in the case of the lungs we examine every part of the surface of the chest by percussion and auscultation, so in the case of the nervous system we investigate every function—the power of motion in every part of the body, the accuracy of sensation, the perfection of the special senses, and the integrity of the intellect and emotions. Having ascertained the seat of the lesion, we may, from our knowledge of the diseases that affect certain localities, make a complete diagnosis; on the other hand, we may sometimes, from the nature of the attack, or from points in the patient's history, be enabled to recognise the nature of the lesion, when its exact localisation is still uncertain.

Before proceeding to the systematic description of the diseases of the nervous system, something must be said of its general anatomy and of the clinical methods of investigating the symptoms which its diseases produce.

GENERAL ANATOMY

The organs of the nervous system are made up of nerve-cells and nerve-fibres, united together by connective tissue, a special form of which in the brain and spinal cord is known as *neuroglia*. Each nerve-fibre is associated with a nerve-cell, so as to form a separate structure, the *neuron;* and practically the whole nervous system is made up of neurons, of which at least some are motor and others sensory. A neuron consists of a cell-body, from which proceeds one long fibril, the axon, and other shorter processes, dendrites. The *cell-body* is variable in shape and size, has a large nucleus and nucleolus; and the protoplasm presents a mottled appearance, due to Nissl's granules. The axon, or axis-cylinder process, formerly known as the axis-cylinder, is smooth, uniform in diameter, runs for long distances as a nerve-fibre, and joins with others to form nerve-trunks; it gives off branches from time to time called *collaterals*, and ends in tufts of fibrils called *arborisations*. The terminations and collaterals are distributed around other cell-bodies and dendrites. The *dendrites*, or protoplasmic processes, are often rough on the surface, break up into branches close to the cell-body, and these into minuter ramifications which do not anastomose.

How exactly the axon-terminals of one neuron are connected with the dendrites of another is still a question. A direct union of protoplasm has been generally denied. Sherrington supposes a receptive substance exists, which he calls *synapse*.

An important fact from the pathological point of view is that the nutrition of every part of the neuron, whatever its length—and some reach from the cerebral cortex to the lower end of the spinal cord—depends upon its connection with the cell-body and the integrity of the nucleus.

The motor functions of the nervous system are served by two groups of *motor neurons*. The *upper* or *central motor* neurons have their cell-bodies situated in the cortical motor area of the brain, forming the well-known pyramidal cells; they have a few dendrites in the immediate neighbourhood, and they send long axis-cylinder processes downwards which form the pyramidal tract, cross the middle line (decussation), occupy the crossed pyramidal tracts of the spinal cord and terminate by arborisations which interlace with the dendrites of the neurons of the other group. This comprises the *lower* or *peripheral motor* neurons; their cell-bodies are situate in the anterior cornua of the spinal cord and the corresponding nuclei of the medulla oblongata and pons, their axis-cylinder processes pass out into the motor-nerves, and their final tufts or arborisations come into relation with the end-plates of the muscles.

The groups of sensory neurons are three in number. Of the *lowest* or *peripheral* neurons (*protoneurons*) the cell-body is situate in the ganglia on the sensory roots of the nerves. They have an axis-cylinder process, which after a short distance divides into two

branches. One of these goes downwards in the nerve-trunk to be connected with the skin and muscle-spindles, and is regarded by some, not as an axis-cylinder, but as a protoplasmic process or

FIG. 14

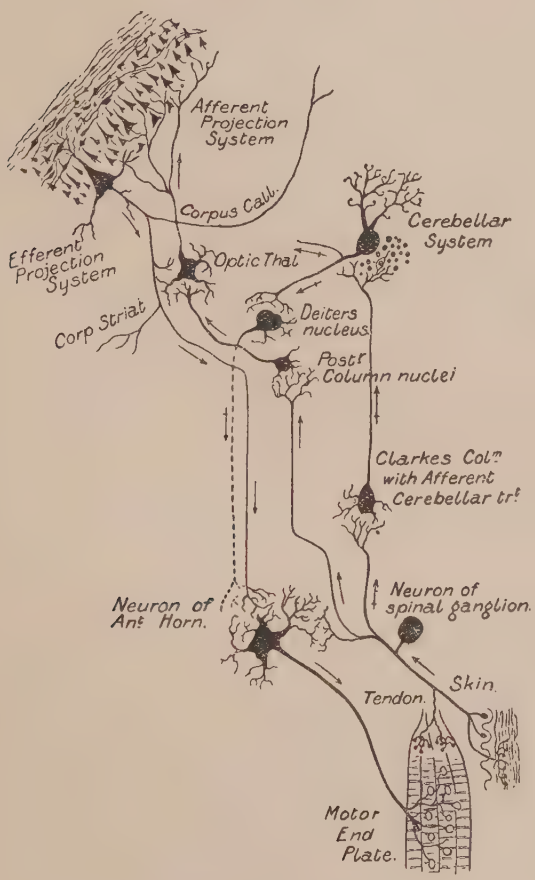


Diagram illustrating the afferent, efferent and association systems of neurons. (*After Mott.*)

dendrite. The other branch passes upwards into the cord, where again it divides, one branch passing downwards, the other upwards. Both send off collaterals, some of which surround the cell-bodies of the lower motor neurons in the anterior cornua. Others surround the large cells in Clarke's column, and these cells are *secondary* neurons, of which the axons pass up in the cerebellar tract of the same side. Other collaterals of the lowest neurons surround the

cell-bodies of secondary neurons, of which the axons cross the middle line and pass up the cord in the opposite antero-lateral ascending tract. The ascending branches of the main axis-cylinder pass up the spinal cord and come into relation with the *secondary* or *middle* sensory neuron, of which the cell-bodies are situate in the medulla oblongata as the nuclei of the posterior columns, *nucleus gracilis* and *nucleus cuneatus;* while their axis-cylinder processes cross the middle line in the decussation of the fillet, join the processes from the antero-lateral ascending tract, and pass upwards in the direction of the cortex. In the thalamus opticus they terminate round the cell-bodies of a *tertiary* or *upper* sensory group of neurons, of which the axis-cylinder processes pass up to the cerebral cortex, when they come into relation with the cells of the post-central convolution. Some of the sensory axis-cylinder processes pass upwards in the posterior part of the internal capsule.

There are, of course, many other kinds of neuron, commissural or subserving the special senses and other functions.

The general results of lesions of the neurons may now be considered.

Destructive lesions.—The lesion of any neuron causes a degeneration of that portion which is cut off from the nucleus of the cell-body, and hence a lesion of the cell-body itself destroys the whole neuron. Lesion of the upper motor neuron (cortical motor area, or pyramidal tract) causes degenerative changes in the axis-cylinder processes below the lesion (*see* Secondary Degeneration), but leaves the lower neuron intact. Lesion of the lower motor neurons causes degeneration of axis-cylinder processes below the lesion, that is, of the nerve-trunks; and as the muscles with which the neurons are connected depend upon them for their nutrition, they become degenerated or atrophied as well.

Functionally the result of destructive lesions on either motor neuron is paralysis to voluntary impulses of the muscle in which the lower of the two terminates. If the upper neurons in the brain are alone affected, the paralysis is on the opposite side of the body, involves as a rule a large number of muscles, such as the arm or leg, or the whole side of the body, and not single muscles; and spinal reflex action remains undiminished. If the lower neuron is alone affected, the paralysis is on the same side of the body as the lesion: single muscles may be picked out or a whole limb involved, according to the number of neurons diseased, spinal reflex action is abolished, and the nutrition and electrical reactions of the nerves and muscles concerned are lost or impaired.

Irritative lesions of the upper motor neuron in the cortical area cause spasmodic contractions of the muscles with which they are in connection on the opposite side of the body Those of the lower motor neurons are less constant in their results. Some spasms in spinal meningitis and the fibrillary twitchings of progressive muscular atrophy may be due to them.

Lesions of sensory neurons cause various disturbances of sensa-

tion, either of defect amounting to complete loss, or of excess including pain.

Head has made important contributions to the subject of sensation in recognising that three kinds of sensibility are transmitted by the peripheral sensory nerves. They are *epicritic sensibility*, the appreciation of light touches, of slight changes in temperature, of cutaneous localisation, and the discrimination of the points of a pair of compasses; *protopathic sensibility*, the recognition of painful cutaneous stimuli, and the extremes of heat and cold; and *deep sensibility*, the sense of position and movement, and of pressure which, if sufficient, may produce pain. The fibres conveying these sensations are contained in the posterior roots, but in the spinal cord take various courses: peripherally those conveying deep sensibility run with the motor nerves and are distributed to the muscles, joints, bones, and peritoneum.

In lesions of peripheral nerves the loss of epicritic sensibility is observed over an area greater than that of the loss of protopathic sensibility; but the difference is less the nearer the lesion is to the spine (*see* Figs. 18 to 24).

Related to injury of the lower sensory neurons are certain lesions of nutrition of the skin and joints, such as are seen in tabes dorsalis and peripheral neuritis.

CLINICAL EXAMINATION OF THE NERVOUS SYSTEM

MOTOR SYMPTOMS

These consist of *paralysis, spasm* or *convulsion*, and *inco-ordination*.

Paralysis, or loss of power in the muscles, is mostly due to lesions of the nervous system; but it occurs in some diseases of the muscular substance itself, such as pseudo-hypertrophic paralysis. Loss of power may also arise from defective nutrition, such as follows prolonged illness; but it is not then included under the term paralysis. In all forms, the weakness may be of any degree up to complete abolition of movement. The lesser degrees are sometimes called *paresis* or *partial paralysis*. Paralysis in the upper limb may be tested by getting the patient to grasp one's hand with his, by forcibly extending (or flexing) his arm, which he tries to keep flexed (or extended); in the lower limb by his power to stand upright, or to raise the heels from the ground, or to carry a weight on the back, or, while sitting on a chair, to flex or extend the leg against the observer's pressure; or, if in bed, to raise the foot and leg from the bed, or to draw the knee up to the abdomen, or to raise the leg while the observer presses upon the knee; in the abdominal muscles by his power to raise his head from the pillow, or to raise the body from the bed without using the arms, or to cough; in the muscles of the back, by his power to extend the

head while lying on his face. Comparison should be made with the limb of the opposite side, or with the muscles of a healthy individual of the same sex, age, and muscular development. The grasp of the hand can be usefully tested by the *dynamometer*, which, in its usual form, consists of an oval steel ring 5 by 2½ inches, which, when compressed laterally, registers the extent of movement on a dial, scaled to pounds. It can also be used by pulling on the two ends.

Names have been given to special forms of paralysis, such as *hemiplegia*, paralysis of one side of the body; *paraplegia*, paralysis of the legs, or the legs and trunk; *monoplegia*, paralysis of one limb, &c.

Spasms, or *Convulsions*, are morbid involuntary muscular contractions, which may be (1) interrupted, or *clonic* (κλόνος, violent movement); (2) continuous, or *tonic* (τείνω, to stretch).

Clonic convulsions.—In the convulsions of epilepsy, uræmia, puerperal eclampsia, irritant and alkaloidal poisoning, and organic cerebral disease, and in those occurring in infancy and hysteria, there are quickly alternating contraction and relaxation of antagonistic muscles, so that violent to and fro movements of extension and flexion with considerable displacement of the parts are produced.

In less pronounced conditions of the above diseases, and in chorea, spasmodic wryneck, myoclonus, and the spasmodic tics, clonic spasms consist of sharp contractions or twitchings of more isolated muscles with slower relaxation, and with relatively less displacement of parts. In athetosis, which is most marked in the distal extremities of the limbs, the movements are slow, irregular, and often the result of combined action of several muscles.

Almost inseparable from clonic spasms are the movements called *tremors*, or tremblings, in which oscillations of the part occur also from alternate contractions and relaxations of antagonistic muscles: they are slight in extent, producing displacements of the limb which may not exceed a few tenths of an inch; they may occur as often as six or eight times in the second; and they may be regular and uniform, or *rhythmical*, over long periods. These are seen in shivering or *rigor*, whether toxic or hysterical, in nervousness, emotional states, muscular exhaustion, alcoholism, Graves' disease, paralysis agitans, senile weakness, and poisoning by lead or mercury.

In most cases the movements can be checked for a moment by an effort of the will; but in some complaints, the limbs are quiet when the person is at rest, while the attempt to perform any action causes tremors, which, in disseminated sclerosis, are made up of much more extensive movements than in the above instances. The limitation of the tremors to the time of attempted movement has led them to be called *intention tremors*.

Fibrillary tremors or *twitchings* are quick contractions of isolated portions, or fibrillæ of muscles, just visible under the skin, but incapable of moving any part of the limb.

Tonic convulsions.—These are seen in tetanus, tetany, hysteria and

catalepsy, in strychnia-poisoning, in meningitis, and other organic cerebral and spinal diseases, in myotonia congenita, and writer's cramp. The contraction of the muscle is continuous without relaxation over several seconds, minutes, hours, or even days. The part to which the muscle is attached is either not displaced at all, or it is, in varying degrees in different cases. If the contraction persists for weeks or months, it may be called *contracture;* and in very long continued cases structural changes take place in the muscle which hinder recovery.

In some cases of localised paralysis, permanent contraction results in the antagonist muscles.

Inco-ordination.—Muscular movements, especially the more complicated, require an exact adaptation in the contractions of the different muscles concerned. These are not only the muscles primarily needed to effect the movement, but also their antagonists; and, if this adaptation is imperfect, the movement becomes irregular, or disorderly, and *inco-ordination*, or *ataxy*, results. In the legs it may be tested by asking the patient to walk along a straight line, when the gait is found to be staggering, or reeling, or waddling; to turn quickly when walking; or to lift the heels from the ground when standing. In *Romberg's test*, the patient must stand with his feet close together, so as to reduce the base of support, and then close his eyes; a failure in the afferent apparatus for standing or walking is shown by his swaying to and fro, or from side to side, and even losing his balance altogether. Inco-ordination in the arms may be obvious on the patient putting out his hand to seize objects; or slighter degrees may be brought out by the patient shutting his eyes, and then trying to touch his nose with the tip of his forefinger, or to hit a spot on a sheet of paper with a pencil.

SENSORY SYMPTOMS

The sensations of touch, temperature, and pain are not always affected together, and each should be separately examined.

Anæsthesia, or loss of tactile sensibility, can be tested by lightly touching the surface of the skin with a feather, or cotton wool rolled to a fine point, while the patient's eyes are closed. He should be able to say *when* and *where* he is touched. Fallacies are common, and the result must be checked by frequent trials. Some idea of the extent of anæsthesia may be gathered by noting the distance at which two points touching the skin can be recognised as two, or are thought to be only one. In the most sensitive parts they are recognised as two when less than 2 mm. apart; in the least sensitive the distance must be 2 or 3 inches. The *æsthesiometer* is an instrument devised for the accurate measurement of these points; it consists of a graduated bar, on which two points can slide easily, so as to be fixed at a required distance from each other. The following are some of the measurements given by Weber, representing the distances at which the points can be distinguished as two, under normal conditions:

Tip of the tongue, 1·5 mm.; finger-tips, 2 to 3 mm.; the lips, 4 to 5 mm.; the cheeks, and backs of the fingers, 12 mm.; the forehead, 22 mm.; the neck, 34 mm.; the forearm, leg, and dorsum of foot, 40 mm.; the chest, 45 mm.; the back, 60 mm.; the upper arm and thigh, 75 mm. Leube has proposed to move a point along the skin, and observe the least amount of movement that can be recognised as such.

A touch may be distinctly felt, but an unusually long interval may take place between the stimulus and the patient's recognition of it; this is *delayed conduction.*

Hyperæsthesia is the production of pain from impressions commonly painless. **Paræsthesia** and **dysæsthesia** are terms used to designate those modifications of tactile, as well as painful, impressions, which result in "tingling," "pricking," "pins and needles," &c., and also various subjective sensations, as formication and the numb feeling. Sometimes a touch is felt, as if it were two, three, or more (*polyæsthesia*), or it may be felt in another part of the body (*alloæsthesia*), or at the corresponding part of the opposite side of the body (*allochiria*). The last is a part of a defect in the subjective idea of "sidedness," which has been called *dyschiria,* and occurs in three forms: *achiria,* in which the patient, though feeling, cannot say which side is touched; *allochiria,* as defined above; and *synchiria,* in which a touch on one side is stated to be felt on both sides. These errors of sensation as to side are accompanied by corresponding errors in attempted movements. They are mental in origin, and occur almost exclusively in hysteria (E. Jones).

Analgesia means insensibility to pain. It may be tested by pinching, or by pricking with a blunt point, such as the point of a quill pen; or by applying the faradic current with wire brush terminals. *Hyperalgesia* may be used for an excessive sensibility to painful impressions.

Sensibility to pressure on the skin can be tested by weights applied to it, and the minimum variation of the weight that can be recognised must be noted. In health it is about one-twentieth of the total pressure.

Sensibility to temperature may be tested by the application of hot and cold spoons, a spoon being heated by immersion in hot water; or by the application of test-tubes holding hot water and cold water. Sensibility should be tested both to moderate temperatures—between 70° F. and 104° F.—and to the extremes above and below these points. Differences of temperature may be entirely unrecognised by the patient; cold may be taken for heat, or the reverse. There are different nerve-endings in the skin for heat and cold respectively.

Muscular sensibility is commonly tested by asking the patient to distinguish between different weights placed in his hand. It is important that no clue should be given to the sense of sight or the sense of touch; hence the weights should be placed in a bag suspended by a string. One may also test the power of recognising the

position into which the limb is put, and the sensitiveness of the muscles to deep pressure, and to electrical stimulation. In the first of these cases, cutaneous sensation must be eliminated by firmly grasping the limb at the point where it is held; and in the last by the injection of cocaine.

Pain.—Subjective pain on the part of the patient should always be critically investigated; and the observer should ascertain what is its exact seat, its relation to nerve-distribution, the times of its occurrence, whether and how it is determined, by movement, or by feeding, or by other physiological process; whether it is aching, burning, stabbing, shooting, &c., and whether it is persistent or intermittent. It is well known that pain caused by disease in a particular region is often *referred* to the far-off extremity of a nerve which arises near, or has connections with the seat of disease; but this applies no more to disease of the nervous system than to that of other parts. The pain of hip-joint disease may be felt in the knee, and that of pleurisy or pneumonia in the flank or iliac region.

Astereognosis is the inability to recognise by touch the shape, size, and consistence of objects, even though their *presence* is allowed. It has been seen in disease of the brain, in compression of the spinal cord, and in locomotor ataxy. The cerebral lesions associated with this symptom have generally been situate in the middle of the Rolandic area (ascending frontal and parietal convolutions), in the subjacent white matter, or in the corona radiata near the optic thalamus. It may result from loss of one or more of the special forms of tactile sensation above specified, viz., the sense of pressure, pain, or temperature, or the appreciation of extent.

REFLEXES

There are three groups of reflex actions, which are of use in the diagnosis of nervous diseases, (1) the *superficial, cutaneous,* or *cerebro-spinal reflexes,* (2) the *deep, tendon,* or *spinal reflexes,* and (3) the *organic* or *complex reflexes.*

Cutaneous Reflexes.—When the skin of the inner side of the thigh is slightly scratched or stimulated, the cremaster muscle of the same side contracts and draws up the testicle. This is a purely reflex action, depending upon the integrity of an afferent and a motor nerve, and of the nerve-centres in the segment of the spinal cord with which they are connected: it is called the *cremasteric reflex.* There are other parts of the surface where stimulation readily elicits muscular contraction; thus, on stimulating the sole of the foot, the toes are moved and the foot and leg are drawn up by flexion at the ankle and knee; this is called *plantar reflex.* A *glutæal reflex* is obtained by stimulating the surface of the buttock; an *abdominal,* by scratching the abdomen along the outer border of the rectus; an *epigastric,* by irritation over the cartilages of the lower true ribs; a *scapular,* by stroking the skin between the shoulder blades, when the attached muscles contract. These movements may be excessive or

impaired or lost. Since they require the integrity of the reflex arc, they may be abolished by disease of the centre, of the afferent or of the efferent nerve; and it is important to know the segment or segments of the spinal cord to which each *reflex* corresponds. These relations are given in the Table which will be found under "Diseases of the Spinal Cord." (*See* pp. 258–259.)

In spinal lesions cutaneous reflex is exaggerated in the parts corresponding to that part of the cord which is below the lesion; but in hemiplegia from cerebral disease the skin reflex is diminished on the side of the paralysis. From these facts it has been supposed that the skin reflex centre in the spinal segment is controlled by a centre situated in the brain, and this again by a third centre situated in the cortex. A later view holds that the so-called reflex centres in the spinal cord are rather the shortest or lowest arcs; and that the sensory impulses are diffused up the spinal cord, and finally contribute to a reflex arc in the cortical cerebral area, which is not inhibitory to the lower centres. If a transverse lesion occurs in the spinal cord, the afferent stimulation is concentrated in the lower arcs; if the lesion is in the brain, afferent stimuli are widely spread through the spinal cord, but the motor impulse from the cerebral cortex is diminished.

The occurrence of these reflexes is generally accompanied by some sensation, as, *e.g.*, of tickling; they can rarely be elicited by the patient himself; they are never increased, and may be diminished, by increased activity of the other muscles; they are influenced by varying psychical conditions, and distraction of the attention impairs them. They are delayed in cases of delayed sensation.

In the plantar reflex the movements of the toes are not the same under all conditions. In healthy persons generally the toes are flexed towards the sole, and sometimes they do not move at all, but they are never extended. On the other hand, in certain diseases, such as hemiplegia and spinal diseases, in which the pyramidal tracts are sclerosed or even temporarily involved, the toes, especially the great toe, are extended. This is known as *Babinski's sign*. It is not invariably present, even when the tracts are deeply affected; it may occur in cases of functional paralysis; and it is normal in the new-born infant.

In similar conditions—that is, when there is a lesion of the motor tracts—reflex extension of the toes can be caused by two other forms of stimulus, viz.:

Oppenheim's test, which consists in passing the handle of the percussion hammer along the inner border of the tibia; and

Paradoxical reflex of Gordon. In this the patient sits on a chair with his feet on a stool; the observer, placing the hand on the inner side of the tibia, presses the tips of his fingers deeply into the calf muscles. Thus a stimulus applied to the flexor muscles causes extension.

The last two reflexes are probably deep rather than cutaneous reflexes.

Deep Reflexes.—When the leg is hanging freely, with the knee bent at a right angle, and the ligamentum patellæ is sharply struck with the tips of the fingers, the ulnar edge of the hand, the edge of a book, or other similar object, the rectus femoris contracts, and the foot is jerked sharply forward. This has been called the *knee-phenomenon, patellar-tendon reflex, patellar reflex,* or *knee-jerk.* It is generally well obtained in the sitting position, by crossing one leg over the other and striking the upper knee. Often it can be obtained by striking above the patella. When the reaction is slight, the patient should sit on a table, with the legs hanging over the edge, and the knees should be bare. If it is not elicited then, the patient's attention should be distracted, as by getting him to hold the fingers of one hand in those of the other, and to look up to the ceiling while he pulls at his hands (*Jendrassik's reinforcement*). Apart from this, psychical influences have no influence upon this reflex.

When the patient is in bed, the leg may be raised by placing the hand under the knee; or, the patella being pushed down by a finger placed across the top of it, this finger is struck with the fingers of the other hand (*depressed patellar reflex*). When obtained in this way, it is generally regarded as exaggerated.

Some new methods of obtaining the knee-jerk in difficult cases may be mentioned. (1) The patient is directed to take a sudden deep breath at word of command, while staring at the ceiling. The tendon must be struck at the moment of deep breath (Krönig). (2) He should be directed to read quickly and aloud from a large book held in his hands (Rosenbach). (3) The patient is in the recumbent position, and the leg is supported by two handkerchiefs looped, one under the leg, the other under the thigh just above the knee, and the latter is raised so that the knee is slightly bent. One is held by an assistant, the other (and best, the lower one) is held by the operator, who applies the test to the patellar tendon (Guttmann). (4) The patient lies on his side in the position of sleep, with hip and knee gently flexed, and eyes closed (Feix).

Reflexes, similar to the knee-jerk, can sometimes be obtained with the following tendons: tendo Achillis, adductors of the thigh, triceps, biceps, supinator longus, extensors at the wrist, and masseter acting upon the lower jaw.

Ankle-clonus or *foot-clonus* is a similar phenomenon, which occurs in certain spinal and other diseases, but is not, like the knee-jerk, present in health, except in a modified form. To elicit it, the patient should be in bed or in a chair; the leg is lifted with the left hand under the knee, so that the knee is slightly bent, and the foot, held firmly by the toes in the right hand, is sharply bent towards the knee. Immediately the calf muscles contract, but as the pressure on the foot is maintained, they relax, again contract, and so alternately contract and relax for an almost indefinite period, constituting the so-called *clonus.* When the flexion of the foot fails to start the contractions, they may be brought out, while the foot

is flexed, by a tap on the front of the leg (front tap), or on the tendo Achillis. The contractions occur at the rate of about seven in a second. The modification of this phenomenon which occurs in health is the series of rapid alternating movements which can be kept up continuously and without effort when, in the sitting posture, the foot rests upon the ground by the toes only.

Fig. 15

Photograph of a child presenting Kernig's sign. Note the contraction of the hamstring muscles. (*After Turner and Stewart.*)

A *knee-clonus* can be sometimes obtained, either as a result of percussing to get the knee-jerk, or by pushing the patella down towards the tibia, while the leg is extended on a couch.

That the knee-jerk is not a simple spinal reflex from the tendon is shown by the facts, first, that the interval between the stimulus and the movement is much less than that required for a spinal reflex from the skin of the knee; and secondly, that the knee-jerk takes place when the patellar tendon nerves are divided. The explanation commonly adopted is that the stretching of the muscle, which is effected by bending the knee, reflexly increases the tone of the muscle, so that a local stimulation readily excites its contraction. As the tone depends upon the cell-bodies of the lower neuron, the integrity of the reflex arc is necessary for the occurrence of the knee-jerk. In foot-clonus the forcible stretching of the calf muscles not only gives the required tension, but also acts as a local stimulant to set up contraction; and the tension continuing, each contraction

is rapidly succeeded by another. The segments of the cord concerned in the production of knee-jerk are those which correspond to the second, third, and fourth lumbar nerves; of ankle clonus, to the fifth lumbar and first sacral nerves.

Periosteal and joint reflexes are also included among the spinal reflexes, and are produced by striking certain bony prominences and joints. They are inconstant in health, and distraction of the attention is generally necessary.

Paradoxical contraction of Westphal is a slow contraction in a muscle when it is suddenly and passively shortened. Thus, if the foot be bent up towards the knee, the tibialis anticus will after an interval contract, and maintain the foot bent for a time and then slowly relax. Its significance is not known.

Kernig's Sign.—In meningitis and some other conditions it is found that if the thigh be flexed at a right angle with the body, either by placing the patient in a sitting posture or by raising the thigh vertically while the patient is recumbent, it is impossible to extend the leg completely on the thigh, in consequence of the contraction of the hamstring muscles (*see* Fig. 15).

Organic Reflexes.—These are the reflex contractions of muscles or viscera for physiological purposes, as in respiration, deglutition, micturition, defæcation, and seminal ejaculation; and under pathological conditions, in sneezing, coughing, and vomiting. They occur as a result of irritation of sensitive places, and are accompanied by specific sensations. They often require prolonged or cumulative stimulation; and they are subject to individual differences and psychical influences.

CHANGES IN NUTRITION

The nutrition of the tissues is profoundly affected in some diseases of the nervous system, but there is no evidence of separate trophic nerves or neurons. The most marked effects are seen in lesions of nerve-trunks and their centres, *i.e.*, those which injure the lower neurons, motor and sensory. Thus lesions of the anterior cornua (infantile paralysis) or of the nerves (injury, neuritis) are accompanied with marked wasting of muscle, which is not present in lesions of the brain or cord, involving the upper neurons only. Wasting of muscle is first shown by flabbiness; later by actual diminution in size. Its extent can be estimated by measurement, but it must be remembered that subcutaneous fat may completely mask a good deal of wasting, so far as bulk is concerned. Other parts besides the muscles are often involved. The skin in some chronic cases becomes thin, red, and shiny—the "glossy skin" of Paget; erythematous, bullous, and vesicular eruptions (*e.g.*, zona), œdema, whitlows and ulceration of the skin may occur; the finger-ends are pinched, from wasting of the subcutaneous tissue; the growth of hair and nails is retarded; and the nails are brittle. The bones may also suffer in their nutrition, becoming brittle or

breaking easily; and if paralysis occurs in early life, growth of a whole limb may be retarded, so that it is eventually one and a half to two inches shorter than its fellow. In acute cases the temperature of the skin is raised, the vessels dilate, vesicles or bullæ form, and bed sores occur on the slighest irritation or pressure.

ELECTRICAL CONDITIONS

Two forms of electricity are commonly used in medicine for purposes of diagnosis: the *faradic, induced* or *interrupted* current, derived from an induction coil; and the *galvanic, voltaic,* or *continuous* current, derived from several cells, numbering from two to forty or fifty, joined in series—that is, with the positive plate of each in connection with the negative plate of its neighbour. In the former the current is alternately *closed* and *opened* (*made* and *broken*) with great rapidity by the mechanism employed; in the latter the closing and opening of the circuit are commonly effected much more slowly by the hand.

When used to obtain muscular contraction one electrode, the *active electrode,* is placed upon the muscle to be tested; and the other, the *indifferent electrode,* upon the spine or some other part of the body or limb, generally on the cerebral side of the muscle to be tested.

Motor Points.—There are a number of points on the surface of the body where the nerves and muscles are especially accessible to electrical stimulation; and on these points the electrode should be placed, if the maximum contraction of any muscle is required. Thus, for the long head of the triceps there is a point close up to the axilla; for the internal head there is one midway down the arm on the inner side; for the brachialis anticus the point is a little below the middle of the arm, at the inner border of the biceps muscle. These are called Ziemssen's motor points.*

The Faradic Current.—This may be applied by placing the two terminals or *electrodes* at no great distance from one another on the surface of the muscle to be stimulated; or on the nerve which supplies the muscle; or the active electrode may be on the nerve or muscle, and the other on the spine. It is indifferent which electrode is uppermost (nearer the nerve-centres), as the current passes alternately each way. The stronger the current, the greater is the contraction. The faradic current causes contraction by stimulating the nerve-trunks and the nerve-endings in the muscles, but not the muscular substance itself. Consequently, when the nerve is injured or degenerated, or cut off from its nerve-centre (lesion of lower motor neuron), the reaction to faradism is completely lost, on application either to the nerve or to the muscle.

The Galvanic Current.—This is generally applied with the indifferent electrode on the spine and the active electrode on the

* Illustrations of them are given in several works on medicine and medical electricity: Erb, Strümpell, Finlayson, Vierordt, Lewis Jones, Dawson Turner.

muscle or nerve. The results are different according to the reaction of the electrodes—that is, whether the active electrode is negative (kathode), and the indifferent electrode is positive (anode), or *vice versâ*. In the former case the current is said to be *descending, direct*, or *kathodal;* in the latter, *ascending, inverse,* or *anodal.* A galvanic current of moderate strength causes no contraction while the circuit is complete—that is, while the two terminals are continuously applied; but contraction takes place (1) when the circuit is broken, for instance, by lifting the terminal from the muscle, or by a switch in the machine, and (2) when the circuit is completed again. These contractions are partly due to stimulation of the nerve-endings, but partly also to stimulation of the muscular substance itself; and in certain stages of nerve injury and degeneration, when the faradic current fails to elicit any reaction, and the galvanic current applied to the nerve gives no result, the latter current applied to the muscle calls forth contractions the characteristic of which is that they are slower and longer than under normal circumstances.

Polar Reactions.—Some important results have now to be pointed out, which are explained by the facts of electrotonus. It has been already stated that the galvanic current may be direct or inverse, and that the contraction takes place when the current is either closed or opened. This gives four different conditions of contraction: (α) Closing the direct (kathodal) current, (β) opening the direct, (γ) closing the inverse (anodal), and (δ) opening the inverse. These contractions are generally indicated by symbols as follows:

(α) KCC, Kathodal Closure Contraction, meaning the contraction which takes place when the circuit is *closed* with the kathode or negative electrode on the muscle (hence a descending current).

(β) KOC, Kathodal Opening Contraction, or contraction when the current is *opened* with the kathode on the muscle.

(γ) ACC, Anodal Closing Contraction, or contraction when the current is *closed* with the anode or positive electrode on the muscle (hence an ascending current).

(δ) AOC, Anodal Opening Contraction, or contraction when the current is *opened* with the anode on the muscle.

If, in health, the attempt be made to ascertain what is the smallest number of galvanic cells—that is, the strength of battery proportionately—which will cause the four kinds of contractions, it will be found that KCC requires the smallest number, often six or eight of an ordinary battery; ACC requires more; AOC as many or sometimes more than ACC; and KOC is only brought out by a very strong current, or by none at all that can be borne by the patient. Moreover, currents that are required to bring out strong contractions with AC (Anodal Closure) or AO (Anodal Opening) produce tetanic contractions with KC (Kathodal Closure).

The ascending order represented by the following formula should be remembered—

KCC
ACC
AOC
KOC

Or in more detail as follows:

Strength of Current.	Kind of Contraction.
Very weak . . .	KCC only.
Weak	KCC stronger, ACC, and AOC.
Moderate	KC tetanic, AC and AO stronger contractions.
Strong	KC strong tetanus, AC and AO stronger contractions, KOC.
Very strong . . .	KC very strong tetanus, AC tetanus, AO strongest contractions, KOC stronger.

Thus, for the same amount of current strength—

$$KCC > ACC,\ ACC = \text{or} > AOC,\ AOC > KOC.$$

In various forms of degeneration of nerve and muscle, these relations are often altered—KCC requires as many or more cells than ACC, and AOC as many or more than KOC. Thus, instead of the order of excitability being—

KCC
ACC
AOC
KOC

ACC may be equal to KCC, thus:

KCC)
ACC)
AOC
KOC

or ACC may exceed KCC, and KOC may exceed AOC, thus:

ACC
KCC
KOC
AOC

These alterations are described as *qualitative* changes, to distinguish them from simple increase or decrease of susceptibility to the current, which may be called *quantitative* changes. The former are also called *polar* changes.

Reaction of Degeneration.—The most important indication of disease affecting the nerve-nuclei, nerve-trunks, or muscles, is derived from the use of both currents, and is known as the reaction of degeneration, for which the symbol RD is used. *Complete* RD consists of loss of excitability in the nerve to both faradic and galvanic currents; loss of excitability in the muscle to the faradic

current; increased excitability of the muscle to the galvanic current, with *qualitative* or polar changes, and sluggish contractions. It occurs in paralysis from lesions of lower motor neurons, but not in primary disease of the muscle fibres.

In muscles showing RD it is also found that contraction is more easily obtained when the active electrode is on the distal end of the muscle than when on the motor point (*longitudinal reaction*). This fact may be of use in testing old and pronounced cases with much reduced irritability.

In *incomplete* or *partial* forms of RD the reaction of the muscle to galvanism is as just described, but the excitability of the muscle to faradism, and of the nerves to either current, is not lost, but only lowered. A *mixed* form is also described, in which the muscle-contractions to galvanism are neither very sluggish nor very prompt, and ACC may not exceed, or may even be less than, KCC. It occurs when degenerated fibres of muscles or of nerves lie side by side with others which are healthy.

Two other changes in the electrical conditions of muscle are seen in the *myotonic reaction* and the *myasthenic reaction* (*see* Thomsen's Disease, and Myasthenia Gravis).

Effects upon Sensory Nerves.—A galvanic current causes a sensation not only on opening and closing the circuit, but also during the passage of the current, unless the latter is very weak or brief. The effects are generally more pronounced at the kathode, but vary with the relative size of the electrodes and with the nature of the saline with which the electrodes are moistened.

With the interrupted (faradic) current, the individual shocks are felt, but with higher rates of interuption the sensation is more continuous, and produces an effect of numbness when applied directly over a sensory nerve.

Measurement of Current.—In recording the amount of galvanism used for testing the nerves and muscles, it is very desirable that a galvanometer should be used, and that the amount of electricity employed in each operation should be recorded in *milliampères*. The number of cells forms no certain guide to the quantity, as their electromotive force, and the resistance of the tissues through which the current passes, vary much from time to time.

SPECIAL SENSES

The special senses may have to be investigated in any case of nervous disease, as they may give important information as to the condition of the centres.

Hearing may be tested by the voice, the watch, or the tuning-fork. If it be defective, it is essential to know if the fault is in the conducting apparatus, or in the percipient nerve structures. The vibrations of a tuning-fork may be heard through the bones of the skull when ordinary air-conduction by the meatus and

tympanum is impaired or lost. This fact is made use of in the following tests.

Weber's Test.—The foot of a vibrating tuning-fork is applied to the middle of the frontal region. If the sound is heard best on the side of the deaf ear, the deafness is due to impaired air-conduction; if it is heard best on the sound side, the defect on the affected side is in the nerve apparatus; if there is deafness on both sides, and the tuning-fork is heard in the less deaf ear, it is evidence of nerve-deafness in the worse ear.

Rinné's Test.—If in health a vibrating tuning-fork is held on the mastoid process until the sound is no longer heard, and is then placed in front of the ear, the vibrations will be again audible. The result is described as *positive*. If the same takes place with a deaf ear, it shows that the nerve apparatus is at fault. If, after the fork has become inaudible on the mastoid, its vibrations are still inaudible in front of the ear (*negative* result) it shows that the defect is in the conducting apparatus.

In any case the external meatus and membrana tympani should be examined, after removal of wax, if any is present.

Smell.—One nasal orifice may be closed, while with the other the patient is directed to smell such substances as musk, valerian, essential oils, eau de Cologne, or camphor. Ammonia and other pungent substances irritate the nerves of touch rather than those of smell.

Taste may be examined by the application of salt, sugar, and quinine to each side of the tongue alternately.

Vision is affected in several ways in nervous diseases. The size of the pupils should be noted, and the two pupils compared. It should be noted whether they contract to light and during accommodation. The patient's power of vision for near objects and distance should be inquired into, and the presence of myopia, hypermetropia, or astigmatism determined. Inability to see objects closer, while they can be seen at a distance, is due, in old persons, to slow changes in the lens (*presbyopia*); in young persons to paralysis of accommodation, which may occur rapidly, as after diphtheria. *Diplopia* or *double vision* is mostly the result of paralysis of the external ocular muscles. The *field of vision* may have to be tested, as some patients have good central vision, and are quite unaware of important defects in the rest of the visual field. In other cases nothing can be seen in the centre (*central scotoma*), while vision is good peripherally; in others there is blindness in one half of the field, or *hemianopia*, and therefore only half vision (*hemiopia*); in others different extents of field for different colours. These may be accurately ascertained with the help of an instrument, the *perimeter*. More roughly they may be arrived at by asking the patient to look steadily at an object about a foot in front of him, and then bringing other objects gradually within his field, from above, below, and either side, and noting when he can first see them. Each eye should be tested separately with the other closed.

In many nervous diseases, both spinal and cerebral, changes are seen in the fundus of the eye by means of the ophthalmoscope. The most important are inflammation of the optic disc (*optic neuritis* or *papillitis*) and atrophy of the disc (*optic atrophy*). They are described on p. 223. Optic neuritis must be looked for apart from any complaint on the part of the patient, for vision is commonly retained at least in part, and the patient may be quite unaware of anything wrong with the eyes. The field of vision is, however, often found to be smaller than normal. With the highest degrees of neuritis and with atrophy, vision is lost or reduced to the mere perception of light. Among other important conditions revealed by the ophthalmoscope are the *retinal changes of Bright's disease, tubercle of the choroid, disseminated choroiditis* in syphilis, and *embolism of the arteria centralis retinæ.*

EXAMINATION OF CEREBRO-SPINAL FLUID

Valuable information can sometimes be obtained in cases of spinal and cerebral disease by an examination of the cerebro-spinal fluid. This can be obtained by the operation of *lumbar puncture.* A syringe bearing a needle is introduced between the third and fourth lumbar spines at a point half an inch from the middle line in the case of an adult, a quarter of an inch in a child, and in the middle line in an infant. The needle is inserted two centimetres deep in a child, four to six centimetres in an adult, and is directed slightly inwards and upwards. The fluid withdrawn from the subarachnoid space can be centrifuged and examined bacteriologically for suspected micro-organisms, or microscopically for leucocytes and other cell-elements (*cyto-diagnosis*).

DISEASES OF THE NERVES

NEURITIS

Inflammation of the nerves, or *neuritis,* arises from direct injury, such as blows, punctured or lacerated wounds, over-stretching, the pressure of bones in fractures and dislocations, and compression by the action of muscles through which the nerves pass. Inflammation in the neighbourhood of nerves may extend so as to involve them; and this may happen in suppurating joints, in osteo-arthritis, in pleurisy affecting the intercostal nerves, or in cerebral and spinal meningitis. Cold is frequently a determining, if not the actual, cause of neuritis, which is then often called rheumatic; and acute infections, like those of enteric fever, small-pox, influenza and diphtheria, are sometimes followed by it. The last, as well as many

other conditions affecting the system generally, produce, as a rule, a multiple neuritis, and are enumerated under that head (*see* p. 217).

Neuritis may be *interstitial*, affecting mainly the connective tissue; or *parenchymatous*, affecting first the nerve-fibres themselves. In acute neuritis the nerve is red and swollen, there may be small hæmorrhages, and the microscope shows leucocytes infiltrating the sheath and the septa between the bundles of nerve-fibres. If the change is limited to the sheath or perineurium (*perineuritis*) the nerve-fibres may escape any serious lesion; but if it is interstitial they are more likely to be affected, the myelin becoming atrophied, and the axis-cylinders suffering less. Ultimately a new fibrous tissue is developed in the interstitial tissue and sheath.

When the disease begins in the nerve-fibres, the myelin of the white substance of Schwann is broken up into fragments and globules, the nuclei of the internodal cells enlarge and divide, and the protoplasm increases in quantity or becomes granular. The axis-cylinders, at first breaking into segments with the myelin, disappear, while the myelin, becoming less and less, may leave the nerve-tubes nearly empty, containing only here and there nuclei, some finely granular matter, or brownish pigment granules. The nerve-fibres are generally affected unequally.

Secondary Degeneration.—Injuries which cut off nerves from their centres cause important alterations below the lesion. These have been closely studied in animals, and similar changes follow neuritis, as well as direct injury, in man. They consist of a degeneration of the nerve-fibres, and atrophy or degeneration of the muscles supplied by them.

The nerve-degeneration known as "secondary degeneration," or "Wallerian degeneration," from the physiologist who first described it, is very similar in its nature to the parenchymatous inflammation just mentioned; and it may be accompanied by varying degrees of interstitial change. In rabbits the first complete interruption of the myelin and axis-cylinder takes place about the second day after the lesion, but in man somewhat later, probably from the fourth to the eighth day. The change takes place simultaneously along the whole length of nerve below the lesion.

Still later the proximal part of the neuron above the lesion is also affected: the Nissl's granules in the cell-body are broken up into fine particles (*chromatolysis*), the nucleus moves to the periphery of the cell, and the outline of the cell becomes rounded; ultimately the cell and its processes may undergo atrophy.

The muscles in connection with such injured nerves become flabby, and lose bulk (*muscular atrophy, amyotrophy*). The muscular fibres diminish in size, the transverse striation becomes less distinct, and the substance granular. Later, the transverse striation is lost, or replaced by longitudinal striation, and there is an increase of the connective tissue between the fibres.

More or less perfect regeneration of the nerve-fibres may take place, most readily in slight lesions; and this begins at the central end by the growth of new axis-cylinders, which afterwards become covered with myelin.

Symptoms.—Neuritis and resulting degeneration involve the paths of conduction for motor and sensory, trophic and vasomotor impulses.

There is *paralysis,* followed by flabbiness and atrophy of the muscles, often with tenderness to pressure, or on contraction. The cutaneous and deep reflexes cannot be elicited.

The *sensory* disturbances are numbness, tingling, "pins and needles," increased sensitiveness or pain or hyperæsthesia; often accompanied by pain and tenderness of the nerve-trunk concerned. Anæsthesia of different degrees is present; but on account of the overlapping of the cutaneous areas of the sensory branches, the area of anæsthesia is less than what is indicated by the anatomical distribution of the nerves. The area of epicritic loss is larger than that of protopathic loss; while the deeper sensibilities are not affected in lesions of the purely cutaneous nerves.

The *trophic* and *vasomotor* changes which may occur have been described above (*see* p. 207).

In severe lesions involving motor nerves the electrical *reaction of degeneration* occurs (*see* p. 210). Faradic irritability in both nerve and muscle diminishes rapidly, and may disappear by the end of the second week. Galvanic irritability in the nerve disappears in about the same time. But the irritability of the muscle to galvanic currents, though at first less than normal, in a few days becomes excessive, and remains in an exalted condition for some weeks, then gradually falls to the normal, or even, for a short time, below it again. Qualitative or polar changes also generally occur, and complete the reaction of degeneration.

The electrical reactions are somewhat different in the slighter forms of lesion. Thus, the usually early fall of irritability may be delayed some days; or the initial fall of the irritability of muscle to galvanic currents may be absent, and the increase may be delayed some days; or, in very slight cases, the irritability of the nerve to both faradic and galvanic currents may be for a few days increased.

In *acute* forms of neuritis there is more or less pain in the nerve itself, and in the part to which it is distributed. The pain is worse at night, and is increased by movements or positions that cause stretching or pressure on the nerve. If the nerve-trunk can be felt, it may prove to be swollen and tender, and rarely the skin over it is red, or even œdematous. Tingling and hyperæsthesia may also be present. Later on, sensation is often diminished, and the muscles may present twitchings or cramps, at the same time losing power and becoming tender. Ultimately they atrophy and give the reaction of degeneration. Slight constitutional disturbance may accompany the onset, but it soon passes off.

In *chronic* neuritis pain is an early symptom, and constitutional disturbance is absent. Changes in sensibility, atrophy of the muscles, degenerative reaction, glossy skin, and other nutritional defects follow.

Sometimes neuritis spreads upwards, and, reaching a plexus, invades the several nerves proceeding therefrom. This is called *migrating neuritis*. It may extend to the spinal cord, setting up myelitis or meningitis, and to some such process Gowers is inclined to refer the forms of paralysis which follow visceral disease (*e.g.*, cystitis), and which have been known as "reflex paralyses."

The duration of neuritis is very variable; slight cases recover quickly, but others may last weeks or months. In recovery from injury to a nerve, or after reunion of a divided nerve, protopathic sensibility returns before epicritic sensibility. Faradic irritability of nerve and muscle, and the galvanic irritability, again appear about the eighth or ninth week (Erb), and gradually attain the normal. The return of power in the paralysed part generally precedes any decided improvement in the electrical reactions. In severe lesions with extensive atrophy of muscle, RD persists for some time, but after some weeks the irritability of the muscle to galvanism also diminishes, and finally becomes extinct.

Diagnosis.—Neuritis must be recognised by its symptoms—sensory, motor, and trophic—being limited to the distribution of a nerve, which is, at the same time, painful and tender. In its early stage it may simulate rheumatism or ostitis, and, from the pain alone, it may be mistaken for neuralgia, in which, however, anæsthesia and motor paralysis do not occur.

Treatment.—The first indications are to remove the cause, if possible, and keep the affected part at rest in such a way as to avoid all irritation of the nerve. In acute cases the diet should be light, the bowels kept open, and salines may be given. General or local diaphoresis is often useful. Hot fomentations or linseed-meal poultices should be applied to the affected part, or leeches in very severe cases. On the other hand, cold is recommended for traumatic cases. In later stages, counter-irritation by blisters, mustard-plasters, or liniments may be used, if the increased sensitiveness of the skin to these agents be not forgotten (*see* p. 208). Internally, mercury in small doses, aspirin, sodium salicylate, and potassium iodide are the best remedies. For chronic cases, counter-irritation should be employed, and electricity in the form of galvanism. Gowers recommends the anode on the inflamed nerve, or painful spot, and a just perceptible current flowing continuously for ten minutes. High-frequency currents also render good service in chronic cases. He advises the muscles to be left alone, unless much degenerated, when they may be stimulated by gentle friction or by a weak interrupted galvanic current.

MULTIPLE NEURITIS

Ætiology.—Multiple or peripheral neuritis (polyneuritis: disseminated neuritis) is due probably in every instance to intoxication with some chemical substance, or with the virus of some infectious disease (toxin).

Alcohol, arsenic, and lead are common causes; less frequently copper, bisulphide of carbon, carbonic oxide, and ergot. Of infectious diseases it is especially liable to follow diphtheria, but it occurs also after simple pharyngitis, scarlatina, rubella, measles, small-pox, typhus, enteric fever, tuberculosis, cerebro-spinal fever, influenza, mumps, cholera, malaria, syphilis, gonorrhœa, pneumonia, septicæmia, puerperal conditions, rheumatism, gout, and diabetes. It forms part of leprosy and of the endemic disease beri-beri; and is probably the explanation of many cases called acute ascending paralysis, or Landry's paralysis. Severe cold, damp, and fatigue are sometimes immediate antecedents, but their mode of influence is uncertain.

Its relations to age, sex, occupation, or climate, &c., are determined in each case by the disease or agent which has induced it. It will be sufficient here to state that as a result of alcoholism it is much more frequent in women than in men, and occurs mostly in the middle period of life.

Morbid Anatomy.—The changes in the nerves have been already described—they may be interstitial or parenchymatous. Interstitial lesions are more marked in the larger and medium-sized nerve-trunks, and parenchymatous lesions in the peripheral parts. The changes are slighter as one approaches the spinal cord, and the anterior roots are usually normal. The spinal cord has been generally found healthy; but fragmentation and disappearance of Nissl's granules in the cells of the anterior cornua have been observed in some alcoholic cases (W. K. Hunter). The muscles are degenerated or atrophic.

Symptoms.—The clinical picture of multiple neuritis varies somewhat with the cause in operation. I have already described the neuritis of beri-beri (p. 108), and that of diphtheria (p. 123).

Alcoholic neuritis.—This is the most familiar form: it is generally slow in its development, and patients may have some of the earlier signs weeks or months before consulting a medical man. The first symptoms to be noticed are generally tingling or "pins and needles," or numbness in the fingers and toes; the sensation of "dead fingers," produced by vasomotor spasm; and muscular cramps, especially in the calves. These abnormal sensations spread gradually to the hands and feet, and then to the forearms and legs. In acute cases there may be constitutional disturbance, with elevation of the temperature to 103° or 104°; but often the symptoms are not prominent enough to lead to an examination. After a shorter or longer time the limbs become weak. The patient may, for a time,

get about his ordinary occupations, but at last has to take to his bed. The paralysis affects the extensors more than the flexors of the limbs, so that the patient is unable to extend the hand, and the toes are pointed as he lies in bed (drop-wrist and drop-foot). While he can yet walk, he has the high-stepping gait already described under beri-beri. The interossei and other muscles are weakened; and, in marked cases, the diaphragm and the vocal cords are paralysed, so that breathing is difficult, and the voice and cough are weakened or abolished, and collapse of the lung may take place (*see* p. 244). A quick pulse may accompany the laryngeal paralysis, from neuritis of the vagus. The facial muscles may also be affected.

The weakened muscles quickly atrophy; and the change is early noticed in the anterior tibial muscles, in the extensor brevis digitorum, the calf muscles, and the interossei of the hand.

The electrical conditions of the muscles are also altered. The reaction to faradism is diminished or lost, but the effect of the galvanic current varies. Sometimes there are well-marked polar changes, so that complete reaction of degeneration is present. Sometimes there is only a quantitative change.

Sensory symptoms vary considerably—anæsthesia is generally limited to the lower parts of the limbs, but there may be hyperæsthesia. "Pins and needles" or severe gnawing or burning pains belong especially to the early stages. The nerve-trunks are often tender, or their compression may cause "pins and needles" or "deadness"; but the most constant is *tenderness of the muscles* to pressure, a condition which lasts for months, even in advanced stages of atrophy; it is usually well marked in the muscles of the calf. The reflexes are generally lost, but are sometimes exaggerated at first. The bladder and rectum are generally unaffected, but the excretions are often passed in bed in bad alcoholic cases, possibly from the accompanying mental state. As the case progresses the skin undergoes trophic changes. Contraction of the limbs may arise, such as flexion at the elbow and pointing of the foot, and adhesions may form in and about the joints. Bed-sores are less common than in spinal cases. With extreme muscular wasting of the body or limbs, it is often remarkable how the form of the face is preserved.

The paralysis is generally accompanied, and sometimes partly masked, by a peculiar condition of mind, which is most frequent in alcoholic neuritis, but also occurs, as was first observed by Korsakow, in multiple neuritis from other toxic causes (puerperal fever, typhoid, influenza). He therefore called it *Psychosis polyneuritica* or *Cerebropathia psychica toxæmica;* and it is also known as *Korsakow's disease.* The patient suffers from loss of memory for recent events, is ignorant of his whereabouts, has false memory, and describes events which have never happened. Thus he will be unable to tell his name, age, the day of the week, or where he comes from; but, on the other hand, he may say that he has been for a walk, or ride, or

has seen certain friends, in obvious antagonism to facts. There may be a stage of talkativeness, or even delirium; but, in advanced cases, the patients show extreme apathy, and complete indifference to surroundings.

Sometimes the symptoms are marked by *inco-ordination* (ataxia), instead of simple paralysis. This may affect the arms or the legs, and may, in the latter case, closely simulate locomotor ataxy of spinal origin.

Neuritis from septicæmia and other general infections.—The symptoms present a general resemblance to those of alcoholic neuritis; but they are often of less extent, and limited to the lower extremities or to the distal parts of the limbs.

Arsenical neuritis.—This occurs occasionally as a result of continued full doses of arsenical preparations given medicinally. In the year 1900, in Manchester and some other towns in the north of England, a number of cases of neuritis, at first thought to be due to alcohol, were shown to be caused by the accidental impregnation of beer with arsenic in the process of brewing.

The distribution and character of the sensory and motor symptoms are much the same as in alcoholic neuritis, but there are in the arsenical form greater cutaneous hyperæsthesia, more frequent affection of the facial muscles and lower intercostal muscles; earlier atrophy, more frequent inco-ordination, and more rapid progress (J. Bury), and extreme sensitiveness of the muscles to pressure (Reynolds).

Lead paralysis.—The characteristic feature of lead neuritis is the early affection of the upper extremities, to which indeed the lesions may be confined. The extensors of the hands are paralysed, and there is consequently "dropped hand" or "dropped wrist." If the arms are held out with the forearms pronated, the hands hang down, and the patient is unable either to raise them or to extend the fingers. If the hand and the first phalanges are supported in the horizontal position, the remaining phalanges can be extended, showing that the lumbricales and interossei are still active. Indeed, the paralysis is often confined to the extensors of the fingers, the lower two extensors of the thumb, and the extensors of the wrist. The extensor ossis metacarpi pollicis and the supinator longus generally escape. The failure of extension is most marked in the little finger, least in the forefinger. After a time the muscles of the back of the arm waste, and a peculiar prominence forms on the back of the wrist, due to a backward displacement of the bones of the carpus, and possibly to a distension of their synovial sacs. Examination with the battery shows reaction of degeneration; faradism applied to nerve or muscle gives no result, and if applied to the extensor muscles it commonly acts through these and causes contraction of the flexors. With the galvanic current there is increased contraction of the muscles, and KCC is greater than ACC.

Sometimes other muscles of the arm are affected, especially the

deltoid, the biceps, the brachialis anticus, and the supinator longus; in the legs, the long extensor of the toes and the peronei. Quite rarely there is weakness without wasting in the upper arms or thighs, or a universal loss of power. The interossei and small muscles of the thumb and little finger may be also paralysed in lead-poisoning, but, according to Gowers, they are more frequently affected with a form in which wasting and weakness come on simultaneously (primary atrophic) than with the above described lesion, where weakness comes on first (degenerative). In this primary atrophic form the reactions to the galvanic and faradic currents are in proportion to the degrees of wasting, as is the case in progressive muscular atrophy. In Australia it has been observed that children poisoned by lead had the legs paralysed (foot-drop) before the arms.

Sensory symptoms are not commonly present with lead paralysis, but there may be darting pains, slight anæsthesia, or tremor. And independent of paralysis, chronic lead-poisoning may cause dull aching pains in the muscles or joints, often with tenderness in the muscles, and tingling and irregular anæsthesia in the limbs. These are not unlike the sensory symptoms of alcoholic paralysis, and are probably due to neuritis.

Associated Conditions.—As neuritis is frequently caused by poisons from without, such as alcohol, arsenic, and lead, its symptoms are often associated with those due to the particular poison concerned: thus, in alcoholic cases, cirrhosis of the liver may be present; in cases due to lead, the characteristic blue line, with anæmia, and possibly other symptoms described under "lead poisoning"; in arsenical cases, various lesions of the skin, viz., pigmentation, keratosis or hypertrophy of the epidermis, erythemata and herpes zoster. Cardiac failure with œdema occurs with the neuritis of beri-beri, and may be present in arsenical, alcoholic, and diphtherial cases.

Diagnosis.—Extensor paralysis of the arms and legs, with wasting and tenderness of the muscles, is a characteristic feature of pronounced cases. In less advanced conditions the legs may be alone affected, and in some cases severe pains in the limbs, not localised to the joints and not having the lightning-like character of those of tabes dorsalis, persist for a long time before muscular power is lost. If the legs are alone affected there may be a resemblance to a lumbar *myelitis*, or to a transverse dorsal myelitis in which the reflexes are abolished; but in either of these cases the sensory loss is much more complete than is common in neuritis, and the functions of the bladder are gravely disturbed. When the motor loss is considerable and the sensory but little, the case may have to be distinguished from *acute ascending*, or *Landry's paralysis;* and this is often difficult, seeing that there is good reason to believe that many published cases of Landry's paralysis have been really due to multiple peripheral neuritis. The combination of sensory symptoms with muscular atrophy and paralysis in all

four limbs, especially if the face be involved as well, helps to distinguish multiple neuritis from the spinal paralysis due to disease of the anterior cornua, whether acute or chronic. The ataxic cases are to be distinguished from tabes dorsalis by tenderness of the calf-muscles; by a gait in which the dropping of the foot is noticeable, whereas the foot is kept well up in tabes; and by the absence of Argyll-Robertson pupil.

Prognosis.—Alcoholic cases may be fatal, either in an early stage, where the cause is not promptly removed; or after months, from emaciation or bed-sores, or phthisis, or cirrhosis of the liver. In many cases the symptoms develop rapidly in the first six or eight weeks, and then the condition of the patient may remain stationary for months, or very slowly improve. After years recovery may be only partial. But in alcoholic and in other cases where the disease is not too far advanced, and the cause can be completely removed, the prognosis is more favourable, recovery taking place slowly in the course of from two to six months.

Treatment.—If any external poison such as alcohol, lead, or arsenic is the cause, it must be henceforth kept from the patient. Complete rest is desirable, and plenty of food should be supplied. In cases due to syphilis, sodium iodide and mercury should be given, and sodium salicylate is recommended for cases following cold. Subcutaneous injections of strychnine have been used with advantage, the doses employed being from $\frac{1}{80}$ to $\frac{1}{20}$ grain two or three times a day. Digitalis may also be used when cardiac symptoms are present. Pains may be relieved by Indian hemp, belladonna, or morphia, by the application of chloroform locally, or by wrapping the limb in cotton wool. As long as the nerves are painful, galvanism by a continuous current is the only form of electricity that should be used; later, when pain has subsided, interrupted currents of electricity and massage may be employed. Care must be taken to prevent contraction of the limbs.

NEUROMA

Growths in the nerves may consist of nerve tissue (true neuroma), or of the same tissues that form tumours in other parts (false neuroma). The former consists of medullated or non-medullated fibres, with varying amounts of connective tissue between the fibres; multiple neuromata commonly consist of this variety. On the other hand, a false neuroma is mostly single, and may be a sarcoma, myxoma, carcinoma, syphilitic gumma, especially in the cranial nerves, glioma very rarely, or, most commonly of all, a fibroma. The subcutaneous ends of the sensory nerves are sometimes enlarged into minute tumours, which are visible as small nodules, and which may be very painful (cutaneous neuromata, tubercula dolorosa). Another form is *plexiform neuroma*, in which nodular, tortuous, interlacing cords are mixed up with much connective tissue. This

form commonly begins in fœtal life; and multiple neuromata are sometimes hereditary, and are said to be associated with a neurotic disposition. Injury from wounds and punctures causes neuromata, as in the case of the painful bulbous ends which form after amputation.

The **Symptoms** are pain, anæsthesia, numbness, and formication in the distribution of the nerve, and paralysis of muscles supplied by it, or more commonly reflex spasms in adjacent or even distant muscles. The tumour may be sometimes felt.

Treatment by medicine is only likely to be successful in syphilitic cases; otherwise the tumours must be removed.

LESIONS OF CRANIAL NERVES

OLFACTORY NERVE

A diminution or loss of the sense of smell (*anosmia*) arises from altered conditions of the nasal mucous membrane, such as excessive dryness, or coryza; and in affections of the base of the skull, involving the olfactory bulbs, such as injury, tumours, caries of the bone, and meningitis. It sometimes occurs in tabes dorsalis, and is not uncommon in hysteria, as a part of hysterical hemianæsthesia. It has sometimes occurred after excessive stimulation of the olfactory nerve by strong odours. It should be remembered that loss of smell may affect the power of appreciating flavours, which really requires the combined action of the sense of taste and the sense of smell through the posterior nares.

Excessive sensibility to odours (*hyperosmia*) is noticed in hysteria and insanity; and morbid subjective sensations occur in the insane, and sometimes as an aura in epilepsy.

The primary cause of these defects must be treated, if possible. In anosmia galvanism may be tried, the positive electrode to the mastoid, the negative to the nasal bones. Strümpell recommends painting with a one per cent. solution of strychnine nitrate in olive oil.

OPTIC NERVE

The optic nerve, chiasma, and optic tract, form part of a neuronic system of visual nerves corresponding in its arrangement with the system of neurons of ordinary sensation. The *lower* or *peripheral neurons* are the bipolar cells of the retina in immediate connection with the rods and cones, ending centrally by arborisations round the ganglion cells of the retina. The *second system* consists of these ganglion cells whose axons form the bulk of the optic nerve and terminate centrally in the external geniculate body, in the pulvinar of the optic thalamus, and in the superior corpus

quadrigeminum. The cell-bodies of the *upper neurons* are mainly in the external geniculate body, and their axons extend centrally in the optic radiations of Gratiolet to the cortical gray matter about the calcarine fissure. Other upper neurons are in the pulvinar, and their axons extend to the greater part of the occipital lobe; and other neurons arising in the corpus quadrigeminum come into relation with the nuclei of the ocular muscles.

Optic Nerve.—Inflammation of the nerve, or *optic neuritis*, to which reference has already been made (p. 213), is recognised by the following changes seen with the ophthalmoscope: In the early stages the disc is more vascular, red instead of pink in colour; the edge is blurred, indistinct, and changes its thin sharp outline for a broad violet or purple zone; the retinal veins become tortuous. In later stages the disc looks larger than normal, the surface is red or purplish-red in colour, with an appearance of radial striation; the veins, as they lie on the retina, are markedly tortuous, full, and dark, whereas on the disc itself they are partly or entirely concealed by effusion; the arteries are thin. Subsequently the disc becomes more prominent; it may be mottled white and red by effusion of lymph and blood respectively, which quite conceal the retinal vessels, while the disc vessels are increased in number and size; or the effusion may be so great as to form a projecting button of gray colour, from which proceed the very thin retinal arteries and full tortuous veins. The projection of the papilla into the cavity of the eyeball may be measured in terms of diopters. After three or four months the lymph is gradually absorbed, and atrophy takes place, producing a small disc of bluish-white colour, with small retinal vessels (*optic atrophy*). It arises from a variety of causes, and these are: (1) in the orbit—orbital tumours, aneurysms of the ophthalmic artery, and rheumatic inflammation; (2) in the cranial cavity—cerebral, cerebellar, and meningeal tumours of all sizes and in all situations, acute and chronic meningitis, chronic hydrocephalus, and thrombosis of the cerebral sinuses; (3) general causes—malignant endocarditis, Bright's disease, leuchæmia, chlorosis, lead-poisoning, enteric fever, and some other conditions. When it is due to orbital disease, it occurs only on the same side as the affected orbit, and is double only when both orbits are diseased: intra-cranial lesions, whether unilateral, median, or multiple, as well as altered conditions of the blood, cause, with rare exceptions, a *double optic neuritis*. But when the lesion is unilateral, the changes in the discs are often unequal in extent, and differ in date of onset.

Retrobulbar neuritis affects the nerve behind the eyeballs, and causes at first no change visible with the ophthalmoscope; but later the disc may become atrophied. It gives rise to rapid blindness, severe headache, or pain in the eyes, aggravated by movements of the eyeballs or pressure upon them in a backward direction. In slighter cases, the condition of the optic nerve fibres is indicated by the fact that the pupil contracts to light at first, but that the

contraction is not maintained with continued exposure. *Axial neuritis,* or neuritis of the central fibres of the nerve, leads to loss of vision in the centre of the field (central scotoma).

Optic atrophy occurs as a late stage of optic neuritis (secondary atrophy), and occasionally as a primary lesion, without any previous neuritis, as in tabes dorsalis. Vision is very greatly reduced, or abolished entirely, by optic atrophy.

Optic Chiasma.—This may be affected by tubercle or syphilitic gumma or meningitis, by the pressure of internal hydrocephalus, or of tumours of the pituitary body, and by hæmorrhage into its substance. Since it contains the decussation of those fibres which pass from the left tract to the nasal half of the right retina, and those which pass from the right tract to the nasal half of the left retina, it follows that its lesions produce paralysis of the *nasal* half of each retina, and a corresponding blindness in the *temporal* half of each visual field ; this forms a *double temporal hemianopia.*

If the lesion extends sufficiently to one side, or forwards in the optic nerve, or backwards in the optic tract, the direct fibres on that side are affected as well, and the vision of that eye is quite lost.

A *double nasal hemianopia,* or blindness as regards the inner half of each field, would result from a separate lesion on each side of the chiasma, involving the direct fibres to the outer half of the retina. It is necessarily very rare.

Optic Tract.—The optic tract contains fibres which pass to the visual centres, partly direct from the outer (temporal) half of the retina of the same side, partly across the chiasma from the inner (nasal) half of the retina of the opposite eye. A lesion of the *left* optic tract paralyses the left half of each retina, and gives blindness in the *right* half of each field ; similarly a lesion of the right optic tract produces blindness in the left half of each visual field. This form of blindness is called *lateral* or *homonymous hemianopia,* in contradistinction to the above-mentioned double nasal and double temporal hemianopia, which are called *heteronymous.*

Lateral hemianopia may be caused not only by lesions of the tract itself, but by diseases of the brain implicating the occipital lobe, and the fibres constituting the neuronic system above described. The dark part of the field may be a complete half, or it may be less ; and this partial hemianopia is more likely to arise from lesions in those posterior situations where the conducting fibres are less closely united together. More positive information as to the position of a lesion causing lateral hemianopia may be obtained from the *hemiopic pupillary reaction* (Wernicke). When a light is thrown upon the blind half of the retina, the pupil contracts if the lesion is posterior to the anterior quadrigeminal bodies ; the pupil is inactive if the lesion involves the anterior quadrigeminal bodies, or the optic tract itself. This is explained by the relation to the oculo-motor nerves of the neurons which enter the anterior (superior) quadrigeminal bodies (*see* p. 223).

A tumour in the occipital lobe may cause lateral hemianopia; and if the occipital lobes are successively or simultaneously affected by any lesion there will be complete blindness, or double lateral hemianopia. Optic neuritis may in some such cases be entirely absent, and it is not a necessary part of lateral hemianopia. A transient hemianopia may occur in cerebral hæmorrhage; and a special form of lateral hemianopia is a striking phenomenon in migraine. In recovery from hemianopia, the field generally clears from centre to periphery, as is common in migraine, or from above or from below; but rarely from periphery to centre.

A patient with lateral hemianopia is likely to keep his head turned towards the dark half of the field, in order to see distinctly things in front of him. If the left side of the field is dark, the right half of each retina is paralysed; if then the head is turned to the left, objects in front of the patient fall upon the left, or normal, half of each retina. In lateral hemianopia, colour vision may also be affected (*hemiachromatopia*).

Another form of visual defect is that known as *crossed amblyopia*, or blindness of one eye due to a lesion on the opposite side of the brain. The lesion then must be behind the tract; and it appears probable that this may be caused by some disease of the cortex of the lower and hinder part of the parietal lobe, the supra-marginal, and the angular convolutions. There is concentric reduction of the field of vision for white light; and the colour field is also reduced, and may be lost entirely. If the apparently sound eye is tested, a much less degree of reduction of the field, both for white light and colour, will also be found. Thus it seems that the visual centre on one side has connections with the eyes of both sides. In some cases of lateral hemianopia, there is concentric reduction of the half field which is not blind, and this reduction is greatest in the field of the eye opposite to the lesion. It is suggested by Gowers that this is due to an extension of the lesion from the centre corresponding to the lateral hemianopia, to that higher centre whose destruction causes crossed amblyopia.

Crossed amblyopia is frequently seen in hysteria, associated with hemianæsthesia.

THIRD, FOURTH, AND SIXTH NERVES

These nerves supply the muscles which move the eyeball, and their lesions are best considered together. The fourth supplies the superior oblique muscle, the sixth the external rectus, and the third the superior rectus, inferior rectus, internal rectus, and inferior oblique, as well as the ciliary muscle, the sphincter of the iris, and the levator palpebræ muscle. The constant association of the eyes together in all their movements leads, when any one muscle is paralysed, to some important motor and visual disorders, by which the paralysis may be recognised. These are: limited movement,

strabismus, secondary deviation of the sound eye, erroneous projection, and diplopia.

Limitation of Movement.—This is in proportion to the amount of paralysis: in extreme cases—for instance, of paralysis of the external rectus—the eye cannot be moved outwards beyond the middle line. After a time contracture of the internal rectus takes place, and the eyeball is turned into the inner canthus.

Strabismus, or squinting, is a want of correspondence of the visual axes. This occurs when the two eyes look at an object which is in the part of the field corresponding to the action of the paralysed muscle. Thus, if the right external rectus is paralysed the right eyeball cannot move outwards; and when an object in the right half of the field is looked at, the left eyeball moves to the right, while the right eyeball remains stationary: the visual axes converge, and so much the more strongly the farther to the right the object is situate. This is called *convergent* strabismus. If the right internal rectus is paralysed, the left eye can follow an object to the left, but the right remains stationary, looking forwards. The visual axes diverge, and so much the more strongly the farther the object is to the left. This is *divergent* strabismus. The divergence of the axis of the affected eye from correspondence with that of the sound eye is called the *primary deviation.*

Secondary Deviation.—If, in the above circumstances, while the sound eye is looking straight at (or fixing) the object, it be covered, and the affected eye be made to fix the object, the sound eye will be moved still farther in the same direction—that is, inwards with paralysis of the opposite external rectus, outwards with paralysis of the opposite internal rectus. This so-called *secondary* deviation is the result of the increased innervation thrown into the paralysed muscle, and this acts with the greater effect upon the unparalysed muscle opposite. If subsequently the affected eye be covered, the sound eye, again fixing the object, returns to the position it formerly occupied.

Erroneous Projection.—We judge of the position of objects in relation to our own bodies by the movement of the eyeball, or rather by the amount of nerve force supplied to the muscles that move it. If the eyes are at rest in the middle of the orbit, we know that an object in the middle of the field is straight in front of the body; if it moves, and we follow it with the eyes, we judge of its new positions by the movement of the eyeballs. If a muscle is paralysed the increased effort to move suggests a greater movement than has really taken place, and so gives the idea that the object is farther in the direction of the movement attempted than it really is. If the patient tries to touch it with his finger he strikes too far in that direction and may miss it entirely. The erroneous projection is always in the direction of the action of the paralysed muscle: outwards, in paralysis of an external rectus; inwards, in paralysis of an internal rectus; upwards, in paralysis of a superior rectus.

Diplopia, or Double Vision.—If the ocular muscle is paralysed, the erroneous projection of one of the images, while the other is normal, accounts for the perception of two images instead of one. This is called diplopia, or double vision; and more particularly *binocular diplopia;* for it requires the use of both eyes, and when one is closed, a single image is alone seen.* Another explanation of diplopia is that the images of the object on the two eyes are formed in parts of the retina which do not correspond. The image seen by the sound eye, which *fixes,* is clear and sharp, and is called the *true* image. The image seen by the paralysed or inactive eye is less distinct, because it does not, like the other, fall upon the yellow spot; it is called the *false* image. There are two forms of binocular diplopia: *Homonymous diplopia,* in which the right-hand image corresponds to the right eye, and the left-hand image to the left eye, is caused by paralysis of the muscles which turn the eye-ball outwards, namely, the external rectus and the two oblique muscles; *Crossed diplopia,* in which the right-hand image belongs to the left eye, and the left-hand image belongs to the right eye, is caused by paralysis of the muscles which turn the eyeball inwards, namely, the internal, superior, and inferior recti muscles. Hence homonymous diplopia occurs with convergent strabismus, and crossed diplopia with divergent strabismus. In paralysis of the lateral muscles during horizontal movements the images are parallel with one another on the same level; but in other movements and with other paralyses they are on different levels, or inclined to one another.

In examining cases of diplopia a piece of coloured glass may be placed over one eye to distinguish the image that corresponds to it.

Diplopia may be diminished or increased by the use of prisms; diminished if the prism is placed with its base in the direction of action of the paralysed muscles; increased if it be placed in the other direction.

The results of paralysis of the individual muscles are given on p. 228, in a tabular form; in Fig. 16 the appearances presented by a vertical rod in different forms of diplopia are shown; and Fig. 17 is a diagram suggested by Dr. Bruce-Ferguson for helping the memory in the diagnosis of these cases. The circle with the prolonged radii represents the left eye seen from the front, or the right eye from behind. The arrow-head at the end of each prolonged radius shows the direction in which the corresponding muscle acts, whether upwards, downwards, outwards (to temporal side), or inwards (to nasal side); that marked "Third Nerve" represents the resultant action of all the muscles supplied by that nerve.

In paralysis of each muscle, the deviation of the eyeball is in the opposite direction. The false image diverges from the true in the same direction as the muscle acts. When this divergence is outwards, or to the temporal side of the eye concerned, the diplopia is

* Diplopia which occurs when only one eye is used is called *monocular diplopia;* it is due to defects in the cornea, lens, or iris.

Muscle Paralysed.	Diplopia.	Half of Field.	Position of Images Relative to one another.	Effect of Movements on Relative Position of Images when looking—	Defect of Movement.	Secondary Deviation, by—	Strabismus on Movement in Direction of Deficiency (column 6)	To avoid Diplopia, Face turned towards–
External rectus.	Homonymous.	Outer.	Parallel and at same height.	Outwards—separation increases.	Outwards.	Opposite internal rectus.	Convergent.	Affected side.
Superior oblique.	Ditto.	Lower.	False lower than true; inclined to it at upper end; appears to be *nearer*.	Downwards and outwards—difference of height is less, inclination is greater. Downwards and inwards—*vice versâ*.	Downwards and outwards.	Opposite superior oblique.	Convergent only below horizonlal plane.	Sound side, head down.
Inferior oblique.	Ditto.	Upper.	False higher than true; inclined to it at lower end.	Upwards and outwards—difference of height is less, inclination is greater. Upwards and inwards—*vice versâ*.	Upwards and outwards.	Opposite inferior oblique.	Convergent only above horizontal plane.	Sound side, head carried high.
Internal rectus.	Crossed.	Inner.	Parallel and at same height.	Inwards—separation increases.	Inwards.	Opposite external rectus.	Divergent.	Sound side.
Superior rectus.	Ditto.	Upper.	False higher than true; slightly inclined to it at lower end.	Upwards and outwards—difference of height is greater, inclination is less. Upwards and inwards—*vice versâ*.	Upwards.	Opposite superior rectus.	Eyeball rotated, upper end outward.	Affected side, head carried high.
Inferior rectus.	Ditto.	Lower.	False lower than true; slightly inclined to it at upper end.	Downwards and outwards—difference of height is greater, inclination is less. Downwards and inwards—*vice versâ*.	Downwards.	Opposite inferior rectus.	Eyeball rotated, upper end inward.	Affected side, head down.

homonymous—the diagram shows that this occurs in paralysis of the inferior oblique, external rectus, and superior oblique. When the

FIG. 16

Homonymous Diplopia.

External Rectus. Superior Oblique. Inferior Oblique.

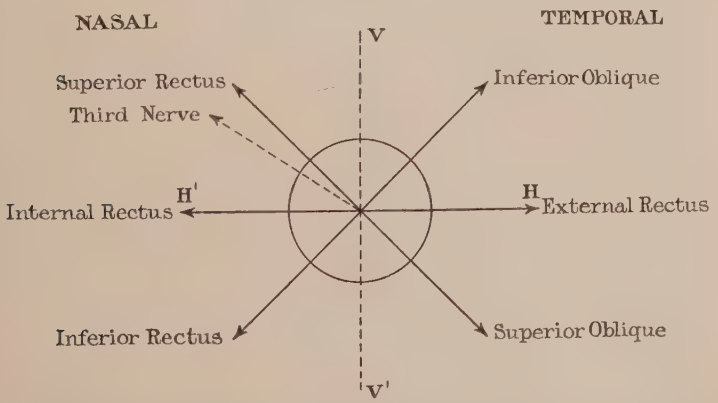
Internal Rectus. Superior Rectus. Inferior Rectus.

Crossed Diplopia.

Images as seen in Diplopia from Isolated Paralysis of Ocular Muscles of the Right Eye. The patient is supposed to be in the position of the Reader. The True Image is White, the False Image Shaded. Two Positions in the Field are given for each form of Paralysis.

divergence is inwards or to the nasal side, the diplopia is *crossed* —the diagram shows that this occurs in paralysis of the superior rectus, internal rectus, and inferior rectus.

FIG. 17



With a vertical object, the inclination of the false image to the true, in paralysis of the two oblique and the superior and inferior recti, is shown by rotating the radius V or V′ towards the line representing the muscle concerned. With a horizontal object the inclination is shown by rotating the radius H or H′ towards the line representing the muscle concerned. The degree of divergence

is proportionate to the divergence of the eyes from one another. The images in paralysis of the internal and external recti are parallel. As an illustration, the superior oblique acts downwards and outwards, and when it is paralysed the eyeball deviates upwards and inwards, the false image is displaced downwards and outwards. With a vertical object the false image is inclined with its lower end away from the true image. With a horizontal object, the false image is inclined with its outer end away from the true image. The diplopia is homonymous.

FOURTH AND SIXTH NERVES ALONE

Lesion of the fourth nerve causes paralysis of the superior oblique muscle, with the results shown in the Table; this nerve is very rarely diseased alone.

Lesion of the sixth nerve causes paralysis of the external rectus.

THIRD NERVE ALONE

This nerve has much wider connections. Its lesions cause paralysis of the superior rectus, inferior rectus, internal rectus, inferior oblique, levator palpebræ, sphincter of the iris, and ciliary muscle. In complete paralysis the eye can only be moved outwards by the external rectus, and downwards and outwards by the superior oblique; at the same time there is *ptosis* or drooping of one upper eyelid from paralysis of the levator palpebræ. There is diplopia in the upper, inner, and lower parts of the field, the false image is higher than the true, and the separation of the images increases with movement inwards. The strabismus is divergent. The eyeball is slightly prominent from the weakness of the recti, and in late cases there is contracture of the external rectus. Accommodation is paralysed, and the pupil is dilated.

Lesions of the third nerve are, however, often partial, and accordingly one or more muscles may escape—for instance, ptosis may be absent; or the levator palpebræ and superior rectus, supplied by the upper division of the nerve, may be affected alone, or escape together; or the external muscles may be alone affected, or the internal (ciliary muscle and pupil contractors) alone.

Internal Ocular Paralysis.—*Cycloplegia* is the loss of the power of accommodation from paralysis of the ciliary muscle. Distant vision is clear, but near vision is blurred and indistinct. It may occur alone, or there may be at the same time an absence of contraction of the pupil which takes place normally with accommodation. The ciliary muscle is supplied by the third nerve by means of fibres which arise in the anterior part of the third nerve nucleus, and pass by the trunk of the nerve to the ciliary ganglion and the ciliary nerves.

Iridoplegia.—Paralysis of the iris occurs in three forms.

Accommodative iridoplegia.—This is a want of contraction during accommodation for near vision.

Reflex iridoplegia.—Loss of pupil light-reflex, or absence of contraction of the pupil to the stimulus of light. The reflex path is through the optic nerve, both optic tracts, probably the corpora quadrigemina, the anterior part of the nucleus of the third nerve behind the centre for accommodation, probably the second fasciculus of the origin of the third nerve, its trunk, the ciliary ganglion with its short root, and the short ciliary nerves; and any sufficient lesion of this path will cause reflex iridoplegia. One illustration of this is the *hemiopic pupillary reaction* (*see* p. 224). In the *Argyll-Robertson pupil* the light reflex is lost, but the pupil contracts with accommodation. This is one of the earliest symptoms of tabes dorsalis, and is then often associated with minute contraction (*myosis*); it is seen also in syphilis and in general paralysis of the insane, but rarely in any other condition.

Loss of the skin or sensory reflex.—On pinching the side of the neck, the pupil of the same side will be noticed to dilate, and the same may happen when the palm of the hand is tickled. The motor path of this reflex is in the cervical sympathetic and the fibres connecting this with the cord at the lowest part of the cervical region. The centre is said to be beneath the corpora quadrigemina. In some diseases involving this path the reflex is absent.

Ophthalmoplegia.—By this term is meant paralysis of the ocular muscles in general. *External ophthalmoplegia* is paralysis of the muscles outside the eyball; *internal ophthalmoplegia*, paralysis of the pupil and ciliary muscle; *total ophthalmoplegia* is paralysis of both internal and external muscles.

The lesions causing ocular paralysis may affect (1) the trunks of the nerves; these are syphilitic and so-called rheumatic inflammations, the pressure of orbital or intracranial growths, or of aneurysms, and rarely tumours of the nerves themselves; (2) the fibres in the brain connecting the nerves with their nuclei; such as hæmorrhage, softening, tumours, and disseminated sclerosis; (3) the nerve-nuclei. Paralyses of ocular nerves occur in connection with some diseases, without our being able to say with certainty what is the seat or the nature of the lesion. Diphtherial paralysis often begins with strabismus and loss of accommodation. In tabes dorsalis, paralysis of the third, fourth, or sixth nerve occurs, which may be permanent or transient. Syphilis may act by causing meningitis or gummatous thickening, and may contribute to the formation of aneurysms, which press upon ocular nerves. Nuclear degenerations also occur with some frequency in syphilitic subjects. Cases of *relapsing* or *recurring paralysis* have been described. They begin with pain in the eye, often with headache and vomiting. These last two or three days, and coincidently, or as they lessen, there is paralysis of several ocular muscles, internal and external. Ptosis is generally present. The paralysis lasts a few days or weeks, and recurs at intervals of

months or a year. The disease lasts from early life to middle age. The cause is obscure.

Ocular Paralysis from Disease of the Nuclei.—Ophthalmoplegia is a common result of disease of the nuclei. Some cases with an acute origin have been recorded, but chronic ophthalmoplegia is much more frequent. It occurs in association with locomotor ataxy, and in syphilitic subjects; and also with progressive muscular atrophy, bulbar paralysis, and general paralysis of the insane: it is gradual in its course, first one or two muscles, then others, and finally, after some years, nearly all the muscles being affected. Ptosis, however, may be absent, and is rarely complete. If present, it gives the patient a sleepy look; the eyeballs are fixed or staring, and sometimes they are slightly prominent. Double vision may be present in the early stages, but often disappears in the course of time. Degenerative changes have been found in the ocular nerve-nuclei. A nuclear lesion is certainly present if the internal muscles are alone paralysed in both eyes, or if the external muscles are paralysed in both eyes without the internal, or if associated muscles in the two eyes are paralysed at the same time. But if both external and internal muscles are paralysed in both eyes, coarse disease of the base of the brain may be a cause, though nuclear lesions are far more probable.

A lesion on one side of the pons affecting the nucleus of the sixth nerve causes *conjugate deviation* of the eyes to the opposite side. The external rectus is paralysed directly, the internal rectus of the opposite eye is paralysed through commissural fibres between the sixth nerve-nucleus and the opposite third nerve-nucleus, and through the fibres to the internal rectus arising there (*see* Hemiplegia). A lesion of the sixth nerve-nucleus is often accompanied by some facial paralysis from the close proximity of the facial nerve-fibres, which wind round it.

Ptosis.—Besides ptosis due to lesions of the fibres and nucleus of the third nerve, there are other forms: *reflex ptosis,* from irritation of the third nerve, especially by decayed teeth; ptosis from paralysis of the fibres of Müller in the fascia of the orbit, which are supplied by the sympathetic; *congenital ptosis,* probably due to a central defect; ptosis in weakly adults, especially women, which is worse in the morning; ptosis in myasthenia gravis; and, finally, *hysterical ptosis.* This last affects both sides; the head is thrown back, and the frontales may be strongly contracted when the patient tries to look up. But the attempt is accompanied by a spasmodic movement of the orbicularis, which prevents the action of the levator.

Treatment of Ocular Paralysis.—This must depend upon the cause, if it can be ascertained. Syphilitic cases should be treated by potassium iodide and mercury, and the same drugs may be, at least tentatively, given in many other cases of less certain origin. For cases of an inflammatory nature, counter-irritation by a blister behind the ear or leeches to the temple should be tried. If cold

has appeared to be the cause, hot fomentations should be applied, and salicylates may be given internally. Galvanism may be applied by holding the well-wetted electrode in one hand and applying the forefinger of that hand to the closed eyelid, opposite the paralysed muscle; the other electrode is on the patient's neck (Buzzard). Diplopia may be relieved by the use of a prism, which should not be strong enough to fuse the images, but only to approximate them, so that muscular efforts may be encouraged. For ptosis in weakly individuals, tonics, such as iron, quinine, and strychnia, are required, as well as local stimulants. Similar remedies, and especially stronger stimulants, such as blisters to the temples, and faradism, should be used in the hysterical form; if one eye only is affected, the other eye should be bound up.

FIFTH NERVE

The fifth, or trigeminal nerve, has both sensory and motor functions, represented by a large sensory and a small motor root. The sensory root enters the Gasserian ganglion, and in front of it divides into its first, second, and third divisions; the third division is joined by the motor root. The first division receives a branch from the sympathetic, which goes to the dilating fibres of the iris; the second division is connected with the spheno-palatine ganglion; the third with the otic ganglion and the chorda tympani nerve. The third division is called lingual gustatory, but it is probable that all taste-fibres pass into the chorda tympani, by which they reach the facial nerve, travel with this to the geniculate ganglion, and pass thence by the pars intermedia to the pons Varolii.

The fibres of the fifth nerve may be injured in any part of its course. In the pons Varolii its origin may suffer from tumours or hæmorrhage; its trunk may be affected by tumours or meningitis at the base of the brain; in front of the Gasserian ganglion the first division is liable to pressure from the tumours about the cavernous sinus, or aneurysms or cellulitis in the orbit; the second and third divisions may be injured by growths in the spheno-palatine fissure. Injuries to the mouth or nose may involve various branches of the second and third divisions, and neuritis of the same may be caused by neighbouring inflammations.

Symptoms.—These must depend on the position of the lesion, and whether it involves the fibres of sensation, taste, or motion. If the *sensory* fibres are involved, the result is anæsthesia of the face, corresponding to the distribution of the nerve; for the first division, anæsthesia of the forehead and anterior part of the scalp, the upper eyelid, and bridge and tip of the nose; for the second division, the malar bone, the cheek, the lower eyelid, the side of the nose, the upper lip, upper teeth, upper part of the pharynx, tonsils, soft palate, uvula, and roof of the mouth; for the third division, the greater part of the temple, the upper and front part of the ear, the auditory meatus, the lower part of the cheek near

the mouth, the lower lip, the chin, the lower teeth and gums, the tongue, part of the mucous membrane of the mouth, and the salivary glands.

The loss of sensation is often preceded by tingling and numbness, or neuralgic pains, and there may be tender points like those found in ordinary neuralgia; it may be accompanied at first by sensitiveness to pain, but this also is finally lost. The conjunctiva, and the nasal and buccal mucous membrane, are, of course, involved as well as the skin. The nose is insensitive to the stimulus of pungent vapours like ammonia, and though smells are at first perceived well, the sense becomes afterwards blunted. In consequence of the mouth being insensitive on one side, food is not chewed on that side, and a thick fur collects on the tongue for want of the cleaning operation of mastication.

Certain *trophic* changes also occur in lesions of the fifth nerve; the secretions of the mucous membranes are diminished, there may be swelling and ulceration of the gums, and the teeth become loose; if the cheek is bitten it heals slowly. Often the cornea becomes inflamed (*neuro-paralytic keratitis*); this begins on the lower side, with cloudiness, opacity, and ulceration, by which eventually the eye may become perforated and destroyed. It has been attributed to irritation of the insensitive surface by foreign bodies; but also to lesions of the nerve-root, and of the Gasserian ganglion. Herpes zoster occurs especially in connection with the first division (*H. z. ophthalmicus*), and appears to arise from inflammation of the Gasserian ganglion.

Loss of taste or *ageusia* occurs in the anterior two-thirds of the tongue from lesions of the lingual gustatory *below* the junction of the chorda tympani. It has been believed that the taste fibres from the back of the tongue take a circuitous course through the glosso-pharyngeal nerve, its tympanic branch, the lesser petrosal nerve, and otic ganglion, to the third division of the fifth. But observations on the results of removal of the Gasserian ganglion show that though loss of taste may succeed it, this is only temporary; and it is attributed by Cushing to post-operative degeneration and swelling of the lingual nerve. Hence transmission should be by the root of the glosso-pharyngeal; although Gowers noted long ago that there was no case on record showing abolition of taste from disease of the root of the glosso-pharyngeal nerve, and that taste is often lost in the back as well as the front of the tongue from caries of the middle ear. Taste fibres from the soft palate and palatine arches pass by the Vidian nerve and great petrosal to the geniculate body on the facial, and hence presumably by the pars intermedia to the pons.

One-sided loss of taste (*hemiageusia*) may have the following associations: If the lesion is in the lingual nerve, anæsthesia of the tongue with anterior hemiageusia; if in the chorda tympani, anterior hemiageusia alone; if in the facial nerve between its junction with the chorda tympani and the genu, facial paralysis with

loss of taste; if in the root of the fifth nerve proximal to the otic and spheno-palatine ganglia, facial anæsthesia with hemiageusia.

If the *motor* portion of the fifth nerve is involved, which is only likely to happen in lesions near the origin of the nerve, the temporal, masseter, and pterygoid muscles are paralysed. In the first two, this can be detected by placing the hand on the temple or the side of the jaw while the teeth are firmly clenched, when a comparison with the other side will detect the want of contraction. If one external pterygoid is paralysed, the jaw cannot be moved to the opposite side; and if the jaw is depressed it deviates to the paralysed side. No impairment of the action of the mylohyoid and digastric, or of the tensor tympani and tensor palati muscles, can be detected. After a time atrophy of the temporal and masseter muscles may be recognised.

Diagnosis.—The presence of severe pain may give for a time a resemblance to neuralgia, but anæsthesia and loss of taste prove an organic origin. If one or other branch is alone affected the lesion is in front of the Gasserian ganglion; if all the branches, it must be near the origin. The association of other nerve-paralyses, such as those of the ocular nerves, or of the motor tract, may also help to localise. Loss of taste occurs as a part of hemianæsthesia, and of hysteria.

Treatment.—Besides dealing with the cause, where this is possible, we may relieve pain by morphia, cocain, or gelsemium; and anæsthesia may be treated by the application of the faradic wire-brush.

SEVENTH OR FACIAL NERVE

The facial nerve is the converse of the fifth, having its motor largely in excess of its sensory functions. The *pars intermedia*, or nerve of Wrisberg, represents a sensory root, while the geniculate ganglion corresponds to a spinal ganglion, and the peripheral filaments are distributed to the interior of the auricle, and the external auditory meatus. The pathological interest chiefly centres in its motor fibres, and the tortuous course of the nerve from the pons through a bony canal to its distribution on the face renders it especially liable to inflammation and compression. Paralysis of the facial muscles, indeed, may be caused by lesions, not only of the facial nerve itself, and of the nerve-nucleus, but also of the facial portion of the cortical centres, and of the fibres which connect this with the nucleus. These last, supra-nuclear, lesions cause a limited form of paralysis, which will be described with *hemiplegia*. We have here to do with the more complete nuclear and nerve-trunk paralysis which goes sometimes by the name of *Bell's palsy*.

Causes.—The nucleus and the fibres in the pons may be involved in tumours in that part of the brain, and occasionally the nucleus is degenerated as a part of *labio-glossal paralysis*. Tumours of all kinds at the base of the brain and meningitis may involve the nerve-trunk between the brain and the internal auditory meatus.

In the petrous bone the nerve is liable to injury from otitis, and suppuration of the mastoid cells; and rarely hæmorrhage has compressed the nerve in the aqueduct of Fallopius. On the side of the face the nerve may be injured by blows, or may be involved in cellulitis or parotid growths. The more common cases of facial paralysis often come on after exposure of the face to a draught, as by sitting at a window; but sometimes without any recognisable cause. Such cases are attributed to neuritis, which probably affects the nerve at its emergence from the stylo-mastoid foramen. Cases of facial paralysis have been recorded after zona of the face, when it must be supposed that the neuritis first affecting the branches of the sensory fifth nerve has spread to those of the motor seventh. Facial paralysis is commonly unilateral. Double facial paralysis (*diplegia facialis*) may occur from bilateral disease of the pons, or from otitis, or from syphilitic lesions successively affecting both nerves. Diphtheria, syphilis by its toxins in the secondary stage, and influenza appear also to cause double facial neuritis. An incomplete, yet double, facial paralysis may occur in multiple neuritis.

Symptoms.—In typical cases the muscles of the affected side of the face are more or less paralysed. The most noticeable features are the inability to close the eye, and the distortion of the mouth on attempting to smile or show the teeth. The forehead is not wrinkled on trying to raise the eyelids or look up. The eyelids cannot be brought together, but there is a permanent fissure of a quarter to a third of an inch in width (*lagophthalmos*). When told to close the eye, the patient brings the lids nearly together, and then rolls the eyeball under the upper lid, so that only the sclerotic can be seen. On smiling or showing the teeth, the angle of the mouth is drawn up on the healthy side; but the lips remain in contact on the paralysed side, and a characteristic elongated triangular opening is the result. Moreover, the lips, and the median fossa under the nose, are displaced to the sound side, so that the tongue, if protruded, *seems* to lie nearer the paralysed side, though actually in the middle line. This can be proved by looking at the incisor teeth. The lips cannot be put together for whistling, or blowing, and air escapes irregularly on the paralysed side: the articulation of labial sounds is imperfect in bad cases. The buccinator is paralysed and food collects between the cheek and the gums. The failure of the nasal muscles may be seen in efforts to sniff, when the nostril on the sound side dilates, while the opposite ala is passive. The paralysis of the depressors of the angle of the mouth and of the platysma can also be shown. An important difference exists between the condition in youth and advanced life. The elasticity of the tissues in youth will keep the different parts of the paralysed face in their normal position so perfectly that during rest there is no want of symmetry, and the paralysis may be quite overlooked until the patient speaks or smiles; but in advancing age elasticity is lost, wrinkles multiply, and the parts which are unsupported by muscular

action fall by the action of gravity, and cause a distortion which can be at once recognised. The lower lid falls away from the eyeball, the lachrymal secretion may thence overflow on to the cheek, and the lower angle of the mouth is depressed. The levator palati, formerly said to be innervated by the facial nerve and the sphenopalatine ganglion, is probably supplied by the spinal accessory through the vagus: it is not affected in facial paralysis. Taste may be lost on the affected side of the tongue, if the facial nerve is diseased in its bony canal between the genu and the origin of the chorda tympani nerve. There may be increased sensitiveness to musical notes of low pitch from paralysis of the stapedius and unopposed action of the tensor tympani. Deafness may be present either from co-existing lesions of the auditory nerve or from aural catarrh.

The electrical reactions undergo changes similar to those in other muscles paralysed by lesions of nerve or nucleus. Faradic reactions are diminished or lost; and galvanic reactions, at first excessive, subsequently become diminished with the development of polar changes.

If wasting occurs in late stages it is not obvious, as the muscles are so thin as to contribute very little to the natural fulness of the face.

Course.—Facial paralysis runs a variable course. The so-called rheumatic form (facial neuritis) often develops quite suddenly, and is complete in a few hours. It may gradually recover in a few weeks or months; it may recover only partially; or the face may remain permanently and absolutely paralysed. A partial recovery is often followed by contracture of the paralysed muscles. These are somewhat shortened, the eye is a little closed, and the angle of the mouth is slightly drawn up by the zygomatici; and, if the muscles of the sound side are at rest, the first impression that one gets is that the paralysed side is active and that the sound side is paralysed. This idea is corrected at once when the patient speaks or smiles, or tries to shut the eyes. The contracted side can contract very little more, while the sound side has a wide range of movement. In this condition, also, the affected muscles cannot be moved independently—in closing the eye, the angle of the mouth is raised; in smiling, the eye is partially closed. This is called *secondary over-action*.

Diagnosis.—The recognition of facial paralysis is not difficult. The important point is generally to distinguish the seat of the lesion. In facial paralysis of *cerebral* or *supra-nuclear* origin, the muscles of the lower part of the face are mostly affected, those of the upper part very little; the wrinkling of the forehead is slight, and the eye can always be closed, though not so tightly as on the opposite side, and the eyeball is not rolled up under the upper eyelid as in peripheral paralysis. A smile is less impaired when produced by emotion than when voluntarily attempted, the electrical reactions of the affected muscles are normal, or nearly so, and reflex contractions may be obtained. In a lesion of the facial

nerve-nucleus, the highest lesion causing *peripheral* paralysis, the orbicularis oris escapes, as the nuclear origin of the nerve of this muscle seems to be connected with that of the tongue. A tumour of the pons not infrequently involves the sixth and eighth nerves as well as the seventh, from their close proximity to one another, and disease in the internal auditory meatus must involve the eighth. Lower down the loss of taste will localise the lesion as already indicated (*see* p. 234). Rheumatic neuritis may begin below this section of the nerve, and involve it by extension.

Prognosis.—This is largely dependent on the cause. In facial neuritis an opinion may be formed from the reaction to electrical currents. If this is still normal after a week or ten days, recovery is probable: rapid and complete RD is unfavourable.

Treatment.—When an accessible tumour is the cause its removal should be attempted. Potassium iodide should be given in cases possibly syphilitic. In ordinary rheumatic paralysis, warmth locally applied, counter-irritation by a blister over the mastoid process, and salines with potassium iodide should be employed. Electrical treatment quickens recovery in many cases, and both continuous and interrupted currents may be used. The positive electrode should be placed on the back of the neck, and the other active electrode applied to the main divisions of the nerve. Massage of the facial muscles is also of value. In uncured cases of long standing, where healthy fibres still persist in the facial nerve trunk, the tone and voluntary power of the muscles have been restored, and deformity has been diminished, by dividing the nerve and suturing its distal end to the proximal end of the divided hypoglossal nerve. The atrophy of the lingual muscles, thus deprived of their innervation, is obviated by uniting the distal end of the hypoglossal to a portion of the spinal accessory (Ballance).

EIGHTH OR AUDITORY NERVE

Various cerebral lesions may involve the nuclei of the nerve in the pons, or its higher connections in the brain; the nerve itself may be injured by meningitis, by thickening of the petrous bone, by aneurysms or tumours: and the expansion of the nerve in the labyrinth may be damaged by acute or chronic inflammation, by syphilitic disease, or degenerative changes.

The results of these lesions are: *Deafness: vertigo* (*see* p. 396), and various subjective sounds, especially *tinnitus*. Excessive sensibility to sounds is more often a functional disorder.

Deafness from the above cause is called nervous deafness, and has to be distinguished from loss of hearing due to interference with the conduction of sound through the tympanum and external meatus (*see* p. 211).

A healthy nerve apparatus is shown if with the tuning-fork on the bone the sound is intensified by closing the ear. If the sound cannot be heard with the tuning-fork on the bone, the nerve appa-

ratus is faulty. When the conduction is found to be normal, deafness must be due to a lesion either of the nerve or of the labyrinth. Which of these is more likely to be at fault must be determined by associated symptoms. These may sometimes point to an intracranial lesion, but, as a fact, deafness is not a very common symptom in cerebral cases, unless the trunk of the auditory nerve is directly compressed by a tumour.

Tinnitus Aurium.—This term includes the various subjective sensations of sound, generally of a ringing, rushing, or roaring kind, with which, in their slighter degrees, nearly every one is familiar. It is clearly due to irritation of the auditory nerve-fibres, and may occur in almost any form of ear disease, whether of the external meatus, of the middle ear, of the labyrinth, or of the nerve or nerve-centres. But structural disease of the nerve or nerve-centres is rarely a cause of tinnitus: and while it may be caused by cerumen in the external meatus, and is not unfrequently present in acute and chronic inflammations of the middle ear, it is much more commonly present in a number of general disorders, which probably through the arterial (or venous) circulation cause impressions upon the auditory nerve apparatus, either in the labyrinth or in the brain itself. Thus tinnitus may be caused by large doses of quinine, or of the salicylates, or by nitrite of amyl: and it is often associated with gout, anæmia, chronic heart disease, atheroma, alcoholism, chronic Bright's disease, and high arterial pressure. It occurs also in migraine and in epilepsy. Deafness frequently accompanies tinnitus, both in functional and structural lesions.

Treatment.—The cause of the tinnitus must be first considered. Disease of the external or middle ear may be directly treated, and any general disorder, like anæmia or alcoholism, should be met by appropriate remedies. When the tinnitus seems to be due to the nerve or labyrinth, the following may be tried: Potassium bromide, hydrobromic acid, quinine, sodium salicylate (Gowers) internally: counter irritation by a blister behind the ear, dry cupping at the back of the neck, or the continuous current of electricity.

NINTH OR GLOSSO-PHARYNGEAL NERVE

There is considerable doubt as to the exact anatomy of this nerve. Its nucleus contains cell-bodies like those of motor neurons: and the petrous ganglion contains the cell-bodies of its sensory neurons, and corresponds to the ganglia of the spinal nerves. Its relations to the sense of taste have been mentioned above (*see* p. 234). Its pharyngeal branches join with those of the vagus in the pharyngeal plexus. The nerve is rarely, if ever, affected alone: the nucleus of the nerve is involved in cases of labio-glossal paralysis, and the trunk is likely to be affected by tumours or other lesions of the medulla, in association with the root of the pneumogastric. In dysphagia, globus hystericus, and anæsthesia of the pharynx this nerve may have a share.

TENTH, PNEUMOGASTRIC, OR VAGUS NERVE

This nerve has both motor and sensory fibres, the latter being connected with the ganglia at the base of the skull; while many of its motor fibres are contributed by the accessory portion of the spinal accessory nerve. The vagus is extensively distributed to the pharynx, larynx, lungs, heart, stomach, intestines, and spleen.

It is liable to still more lesions than the other cranial nerves from its great extent and varied course—*e.g.*, lesions of its nuclei from degeneration, softening, or hæmorrhage, generally in association with the adjacent nuclei; of its roots from meningitis, syphilis, tumours, or aneurysm; of the nerve itself from wounds, surgical operations, aneurysms, new growths, or enlarged glands. The last three are frequently causes of difficulty with the pneumogastric in the thorax and the recurrent laryngeals in any part of their course. Diphtherial, alcoholic, and other forms of multiple neuritis also involve the functions of the pneumogastric nerve.

If the *pharyngeal* branches are affected, swallowing is difficult; the food lodges in the pharynx, and small portions of liquids may pass into the larynx and cause choking. If the *laryngeal* branches are diseased, either in the trunk of the nerve itself, or in the recurrent laryngeal nerve, the various forms of paralysis of the vocal cords, and other parts of the larynx, are produced, which are described in the section on Diseases of the Larynx. *Pulmonary* branches are both afferent and efferent; of the afferent fibres, some stimulate, others inhibit, the respiratory centre; and the efferent fibres are said to supply the muscular fibres of the bronchi. But it is only in rare instances that the results of lesions of these fibres are observed clinically. The phenomenon called Cheyne-Stokes respiration, and the spasms of hydrophobia, are probably dependent on changes in the respiratory centres, with which the vagus nucleus must be connected. The *cardiac* fibres have an inhibitory action, and are believed to be involved in cases of alcoholic neuritis, when the pulse may become excessively rapid; a similar acceleration has occurred from local disease of the nerve-trunk. Some curious cases are on record of slowing of the heart from irritation of the vagus by pressure. Lesions of the *gastric* branches seem to have caused in different cases pain, vomiting, or excessive appetite; the vomiting frequently observed in cerebral disease must be due to irritation of these nerves.

Treatment must be conducted on the lines indicated in the case of other nerves. (*See also* Diseases of the Larynx.)

ELEVENTH OR SPINAL ACCESSORY NERVE

The external portion of this nerve arises by a series of roots from the cervical part of the spinal cord, and is really a motor spinal nerve directly connected with the anterior cornua. It is distributed to the sterno-mastoid and trapezius muscles; it is the chief supply

of the former, but the latter is largely innervated by cervical and dorsal nerves.

In addition to cerebral and intracranial lesions, like those which may involve the vagus, the spinal accessory may be injured by caries of the cervical spine, by enlarged glands or abscesses in the neck, or by blows and strains. If the lesion is in the posterior triangle, the sterno-mastoid will of course be spared. Paralysis of the sterno-mastoid is shown by the want of prominence due to contraction of the muscles, and by deficient power of rotation of the head to the opposite side. In paralysis of the trapezius, the natural slope between the neck and the shoulder is converted into a deep hollow, which is exaggerated when the shoulder is raised, as it still can be by the action of the levator anguli scapulæ. The point of the shoulder lies lower than normal, and the angle of the scapula is rotated inwards by the unopposed action of the rhomboids and levator. Elevation of the hand above the head is, however, difficult or impossible, because the trapezius does not fix the scapula for the use of the deltoid, nor does it assist in that rotation, for which the serratus magnus is chiefly employed. If the whole muscle is paralysed, the approximation of the shoulder-blade to the spine is incomplete; but in spinal accessory lesions it is chiefly the upper part between the occiput and the acromion which is affected. With a persisting lesion atrophy and electrical changes naturally follow.

Treatment.—Here we must deal, when possible, with the causative lesion, and with the muscular failure by electrical stimulation, and perhaps massage.

TWELFTH OR HYPOGLOSSAL NERVE

This nerve, like the last, has a purely motor function, supplying the tongue and most of the muscles attached to the hyoid bone. Its lesions are very similar to those of the two nerves last considered. As the two nuclei are so close to the middle line, they are generally affected together, producing bilateral results. Unilateral paralysis may result from disease above the nucleus, between it and the cortical centre in the ascending frontal convolution; and below the nucleus, from meningitis, simple and syphilitic growths, caries of the cervical vertebræ, and tumours, cellulitis, or injuries beneath the jaw. If it is paralysed on one side, the back of the tongue on that side is slightly raised, from loss of the tonic contraction of the hyoglossus muscle. In the mouth it cannot be moved freely to the same side, but when protruded is pushed to the affected side by the contraction of the posterior fibres of the genio-hyoglossus, and by the elongating action of the transversus muscle on the healthy side. In bilateral paralysis the tongue lies motionless in the mouth. Articulation is impaired in proportion to the loss of movement, but very slightly in unilateral disease. Mastication also suffers at the same time. If atrophy supervenes, the tongue shrinks in bulk and

feels flabby, and the mucous membrane is thrown into wrinkles. The position of the lesion is suggested by the associated symptoms. If it is above the nucleus, there may be hemiplegic weakness on the same side as the lingual paralysis, but there will not be atrophy; if below the nucleus, atrophy may ensue, and paralysis of the limbs, if any, will be on the opposite side. If the symptoms are bilateral, as in bulbar paralysis, the lesion is at or near the nuclei, and it is the same if the other lower cranial nerves are involved.

Treatment.—This must follow the causal indications. To galvanise the tongue a spatula may be used with a wooden handle, and insulated by sealing-wax where it passes over the lips.

LESIONS OF SPINAL NERVES

The spinal nerves arise from the spinal cord, each by two roots, an anterior motor and posterior sensory. These nerves contain the lower motor and sensory neurons, the cell-bodies of the former lying in the spinal anterior cornua, and those of the latter in the ganglia of the posterior roots. The portions of the spinal cord with which successive pairs of nerves are connected are called *segments*, and are named after the attached nerves, and not after the vertebræ opposite which each segment lies. The cervical, lumbar, and sacral nerves soon after the junction of their roots unite with one another, and lose themselves in plexuses, from which emerge the named nerve-trunks to the limbs, such as median, ulnar, sciatic, &c. From this it results that in those regions each segment of the cord corresponds to more than one distal nerve-trunk, and each distal nerve-trunk to more than one spinal segment. This fact has rendered unusually difficult the inquiry as to the relation between spinal segments (*i.e.*, nerve-roots) on the one hand, and the individual muscles and the cutaneous areas on the other. These relations have, however, been worked out by several observers (Starr, Thorburn, Head, and others), and the Table and Figures (pp. 258–261), which give these results, will be found of value in connection with diseases both of the nerves and of the spinal cord.

The nerves may be injured or diseased at the roots, or in the plexus or nerve-trunks beyond them. Isolated nerve paralyses in the limbs are more likely to be caused by lesions below the plexuses, while injuries of the plexuses, or nerve-roots, are more liable to be followed by grouped paralyses.

Lesions of the nerve-roots arise in connection with diseases, injuries and tumours of the spinal cord or spinal column, and the symptoms may be combined with those of the central affection: as, for instance, in meningitis, caries, and the degenerative changes of anterior poliomyelitis and tabes dorsalis. Lesions of the spinal nerve-trunks are mostly injuries from pressure, wounds, fractures, and dislocation in the distal parts; from new growths, aneurysms,

and abscesses in the proximal portions near the spinal column. Exposure to cold may set up neuritis in isolated nerves; and the causes of multiple and peripheral neuritis, already enumerated (*see* p. 217), must not be forgotten.

Seeing that the spinal nerves contain both motor and sensory fibres, the symptoms of their disease are both loss of muscular power and anæsthesia, determined by the distribution of the nerve-fibres to muscles and skin respectively. If the lesion is persistent, atrophy and altered electrical reactions (*see* p. 210) of the muscles will ensue, and perhaps trophic changes in the skin.

Treatment.—The results of these lesions which require treatment are paralysis and muscular atrophy on the one hand, and pain or other sensory symptoms on the other. In either case a removable cause, such as pressure by abscess or tumour, should, if possible, be dealt with; or, if neuritis is the presumed cause, salicylates, aspirin, iodide of potassium, or perchloride of mercury may be given internally; and electricity may be employed, the continuous and faradic currents where muscular defects are predominant, the galvanic alone if there is pain. In the latter case also the local application of liniments of belladonna, aconite, turpentine, &c., may be very valuable. *Rest* is also essential for a quick recovery: the arm should be carried in a sling, or if the leg is affected, the patient should lie in bed.

Some of the more important and frequent lesions of the spinal nerves are here shortly described.

PHRENIC NERVE

The fibres of the phrenic nerve are involved in disease of the cervical portion of the spinal cord, such as acute myelitis; occasionally the nerve is injured by wounds in the neck, and it may be pressed upon by tumours in the neck and thorax. It is not infrequently involved in the paralyses of diphtheria, alcoholism, and beri-beri, and in some other cases of multiple neuritis; and it may be affected in lead-poisoning. The characteristic symptom of a bilateral lesion is paralysis of the diaphragm. The breathing is effected solely by the action of the intercostal muscles, and accessory muscles of inspiration; the abdominal wall, instead of advancing during inspiration, is retracted, and it is driven out during expiration. Dyspnœa may be slight when the patient is tranquil, but movement increases the difficulty, and then the over-action of the thoracic walls becomes especially striking. In a less marked stage of paralysis the diaphragm seems to remain in a semi-inspiratory position, not contracting upon the contents of the abdomen, but it resists being drawn up into the chest, so that the abdominal wall is more stationary during respiration. In a bad case a full respiration is impossible, coughing becomes difficult, husky, or noiseless, from inability to fill the chest, and the voice is almost lost. Two

results are likely to follow. One is extensive collapse (*massive collapse*, Pasteur) of one or both lower lobes, more often the left, with dulness, and absent or faint bronchial breathing. The other is impeded circulation in the lower lobes, so that mucus or œdema fluid accumulates which the patient is unable to expel. The danger thus arising is, of course, increased by any bronchitis. Diaphragmatic paralysis, though it sometimes gives the *coup de grâce* in alcoholic and diphtherial paralysis, is not necessarily fatal—it may last some days or weeks, and then gradually clear up.

POSTERIOR THORACIC NERVE

This nerve is sometimes injured, as it lies in the posterior triangle of the neck, by carrying loads on the shoulder. The lesion is thus common in porters, &c., and is nine times more frequent in men than in women; it also arises from cold, and may be seen in anterior poliomyelitis. The paralysis of the serratus magnus which results is distinguished by the position of the scapula. The inferior angle approaches the spine from the unopposed action of the rhomboidei and the levator anguli scapulæ. The arm is with difficulty raised above the horizontal, since complete elevation is largely effected in health by the serratus magnus rotating the lower angle of the scapula forwards. When the arm is moved forwards in the horizontal position, the angle of the scapula projects from the chest, so that the fingers can be placed underneath it; and it approaches the spine at the same time. Cutaneous anæsthesia is, as a rule, absent, but the onset may be accompanied by neuralgic pains. The digitations of the muscle below the axilla may be obviously wasted or inactive, as compared with the other side.

CIRCUMFLEX NERVE

Dislocations of the shoulder, falls or blows on the shoulder, and the pressure of a crutch are the special causes of paralysis of this nerve. In lesions of the brachial plexus, in lead paralysis, and in spinal lesions, it may also be involved. It is rarely caused by cold. The chief symptom is paralysis of the deltoid muscle, so that the arm cannot be raised to the horizontal position; any attempt results in elevation of the shoulder by the trapezius and serratus, while the arm hangs vertically. In old cases atrophy and reaction of degeneration supervene. Cutaneous anæsthesia is often absent, and paralysis of the teres minor, also supplied by the circumflex, cannot generally be recognised. It must be remembered that ankylosis of the shoulder-joint fixes the arm in the same position, and leads to atrophy of the muscle. Passive movement will distinguish between them.

MUSCULO-SPIRAL NERVE

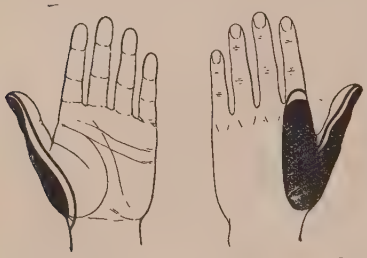
This nerve, from its exposed position as it winds round the humerus, is especially liable to injury from prolonged pressure, from the use of a crutch, or from hanging the arm over the back of a chair during sleep, or from sleeping with the whole weight of the body upon one arm. It may also be injured by fractures and dislocations, and rarely by violent action of the triceps. A partial affection of the branches of the musculo-spiral nerve is the characteristic feature of chronic lead-poisoning. The muscles paralysed by a lesion high up are the extensors of the elbows and wrist, the long extensors of the finger and thumb, and the supinators; but in the more common lesions a little above the elbow, one or more muscles escape, especially the triceps and the supinator longus. With paralysis of the triceps there is inability to extend the forearm on the arm. This must be tested with the arm raised, so as to avoid the action of gravity in extending the forearm. The extensor paralysis of the wrist and fingers is shown by the "dropped wrist" or "dropped hand." If the forearm is extended in pronation, the hand hangs vertically, and cannot be raised, nor can the fingers or thumb be lifted from their pendent position. If the hand be raised and the first phalanges be supported, the middle and terminal phalanges can then be extended by the action of the interossei and lumbricales. The supinator paralysis prevents any movement from the position of complete pronation, but if the forearm be flexed supination will be effected by the biceps. Flexion in semi-pronation is weakened by the absence of the supinator longus, and the characteristic prominence of this muscle in movements of flexion is absent. Flexion of the fingers is considerably weakened by the passive approximation of the ends of the muscles, and a curious prominence forms on the back of the wrist, which is due, either to the thickening of the tendon-sheaths, or to the synovial sacs and carpal bones projecting backwards when unsupported by the extensor tendons. If there is cutaneous anæsthesia, it affects the outer side of the back of the hand, the back of the thumb, and the back of the first phalanges of the fore and middle fingers: epicritic loss is in excess of protopathic loss (*see* Fig. 18).

ULNAR NERVE

This is exposed to wounds and injuries in the arm, and near the wrist, and to injury by dislocations of the shoulder and elbow, and by fractures of the forearm. A neuritis from cold is not common; but the stretching of the nerve at the elbow by extreme flexion of that joint probably sets up neuritis sometimes, especially in those already out of health. The movements affected are flexion of the wrist towards the ulnar side, flexion of the fingers, especially of the first phalanges, with extension of the second and third, adduction of the thumb and the lateral movements of the fingers by the

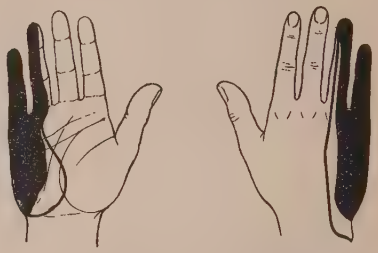
interossei. In old cases the unopposed action of the extensor muscles leads to over-extension of the first phalanges, and flexion of the second and third, producing the claw-like hand (*main en*

FIG. 18



Loss of sensation following a lesion of the radial and external cutaneous branches of the musculo-spiral nerve. The total area of loss is contained within the continuous thick line. The black area represents the protopathic and epicritic loss, the zone between that and the continuous line is the area of epicritic overlap. (*After Head.*)

FIG. 19



Loss of sensation following a lesion of the ulnar nerve. For explanation *see* Fig. 18. (*After Head.*)

griffe). Anæsthesia is variable : its limits are the ulnar part of the hand corresponding to one and a half fingers in front and one (or two) and a half on the back (*see* Figs. 19, 21).

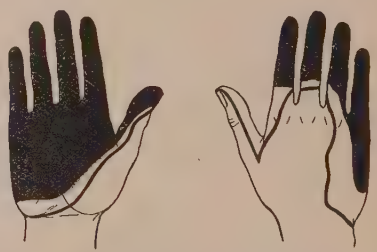
MEDIAN NERVE

This is mostly affected by injuries, occasionally by neuritis. When it is paralysed the forearm cannot be pronated more than half-way, flexion of the wrist takes place towards the ulnar side,

FIG. 20

Shows the area of sensory loss following lesion of the median nerve. For explanation *see* Fig. 18. (*After Head.*)

FIG. 21



Showing the loss of sensation following lesion of the ulnar and median nerves. For explanation *see* Fig. 18. (*After Head.*)

the thumb cannot be flexed or abducted, the second phalanges of the fingers cannot be flexed on the first, nor the third phalanges on the second, except in the case of the third and fourth fingers, in which this is effected by the ulnar half of the flexor profundus. Anæsthesia, if it occurs, affects the radial half of the palm of the

hand, the anterior aspect of the thumb, forefinger, middle finger, and radial side of the ring finger, and the dorsal surfaces of the thumb and the same three fingers (or fore and middle only in some persons) beyond the first joint (*see* Figs. 20, 21).

BRACHIAL PLEXUS

The nerves forming this plexus may be more or less completely involved as a result of injury, growths, or neuritis. This produces paralysis of all the muscles of the hand, arm, and shoulder, as well as anæsthesia of the hand, forearm, and outer side of the upper arm, the inner side being spared because it is in part innervated by intercostal nerves. It is most commonly seen in one of the two forms described below, but there are minor variations in the muscles affected, due probably to differences in the way in which the nerve-roots combine to form the plexus.

Upper Arm Type, or *Erb's Paralysis.*—The following muscles are paralysed: deltoid, biceps, brachialis anticus, supinator longus, and perhaps the supinator brevis, supra-spinatus, and infra-spinatus, and radial extensors of the wrist. Elevation of the arm and flexion and supination of the forearm are impossible. Sensory symptoms in the area of the circumflex and musculo-cutaneous nerves are variable. This is generally stated to be due to a lesion of the fifth and sixth cervical nerves, but W. Harris has shown good reason for believing that the lesion is limited to the fifth nerve. According to him, weakening and partial wasting of the pectoral, latissimus and biceps in addition indicate that the sixth nerve is also affected; and paralysis of the extensors of the fingers probably means the inclusion of the seventh nerve.

Lower Arm Type, or *Klumpke's Paralysis.*—This appears to be due to lesion of the eighth cervical and first dorsal nerves. The small muscles of the hands and the flexors of the fingers are chiefly concerned, and there is anæsthesia up to an inch above the elbow, with loss of epicritic and protopathic sensibilities. In addition there are *oculo-pupillary* symptoms, viz.: Contraction of the pupil on the same side, with diminution of the palpebral fissure.

For cases of upper arm type, Harris has had the fifth root divided and sutured into the sixth and seventh nerve with some success.

SCIATIC NERVE

The most common paralysis in the lower extremity is that due to disease of the sciatic nerve, wholly or in part. It may be from tumours or diseased bone in the pelvis, from dislocations of the hip, from wounds, tumours, or neuroma in the thigh. Neuritis is relatively common, and to this many, if not all, cases of *sciatica* are to be attributed. In a lesion of the sciatic trunk above the upper third of the thigh, the flexors of the leg upon the thigh are involved, in addition to those affected through the two popliteal branches.

EXTERNAL POPLITEAL (PERONEAL) NERVE

This nerve occupies an exposed position near the knee, like that of the ulnar at the elbow. Lesions cause paralysis of the tibialis anticus, the peronei, the long extensors of the toes, and the short

FIG. 22

Front, back, and side views of the leg showing the distribution of the loss of sensibility following injury to the great sciatic nerve. The black area represents the zone of protopathic and epicritic loss, the continuous line the extent of the epicritic overlap. (*After Head.*)

extensor on the dorsum of the foot. The foot hangs down when raised from the ground (foot-drop), and lies extended when the patient is in bed; dorsal flexion of the foot, and of the toes, and abduction of the foot and elevation of its outer border are deficient or impossible. Wasting of the anterior tibial muscles, and of the extensor brevis, can be recognised by comparison with the other leg; in old cases permanent extension of the foot (talipes) is produced mainly by the action of gravity, especially when the patient lies in bed. Anæsthesia affects the outer half of the leg and the dorsum of the foot (*see* Fig. 23).

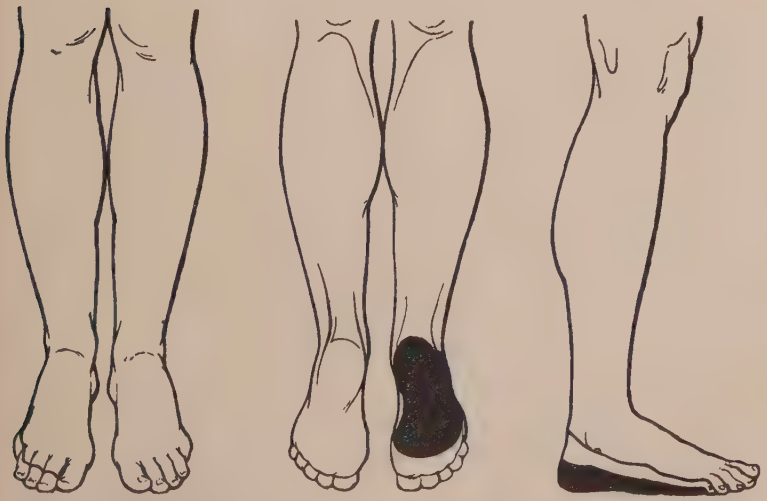
INTERNAL POPLITEAL NERVE

Extension (plantar flexion) of the foot and flexion of the toes cannot be effected. The patient cannot raise himself on his toes, nor can he adduct the foot. In old cases talipes calcaneus may develop, and a kind of claw-foot from over-extension of the proximal phalanges and flexion of the second and third. Anæsthesia

FIG. 23

Areas of protopathic and epicritic loss after lesion of the external popliteal nerve. For explanation *see* Fig. 22. (*After Head.*)

FIG. 24



Areas of protopathic and epicritic loss from lesion of the posterior tibial nerve. For explanation *see* Fig. 22. (*After Head.*)

corresponds to the sole of the foot and its outer border up to the external malleolus (*see* Fig. 24).

SCIATICA

This has long been regarded as a typical neuralgia involving the sciatic nerve, but the fact, so commonly observed, that in old cases the muscles of the lower extremity waste, indicates that there is something more than a purely functional neuralgia; it is, indeed, in a large majority of cases, a genuine neuritis, as proved by the conditions under which it arises; by the accompanying symptoms, anæsthesia, and muscular atrophy; and by the fact that in some cases *post-mortem* neuritis has actually been found.

Ætiology.—The disease is much more common in men than in women, and occurs in the later half of life, especially between the ages of forty and fifty. In many cases no good cause can be shown; in others it arises in connection with gouty and rheumatic tendencies; in a large number of cases it is excited by cold; and syphilis is responsible for some others. It is also set up by mechanical causes such as blows, and long pressure on the nerve, as by the edge of a chair; by fatigue from excessive walking, or otherwise; and by some diseases within and without the pelvis.

Symptoms.—The chief symptom is pain, which is felt in the nerve-trunk or its branches; it comes on either gradually or suddenly, and is aggravated by movement or the attempt to walk, or by anything which causes the nerve to be stretched or pressed upon. To avoid this, the patient holds the leg fixed at the knee when walking, and when lying down the most comfortable position is one of flexion. The pain is most often in the back of the thigh, but may extend down the back of the calf, along the outer side of the leg, and to the sole of the foot; and is often most intense at certain spots—namely, near the posterior iliac spine, at the sciatic notch, about the middle of the thigh, behind the knee, below the head of the fibula, behind the external malleolus, and on the dorsum of the foot. The nerve, too, is tender to pressure, especially at the sciatic notch, along the back of the thigh, and in the external popliteal branch behind the head of the fibula.

The pain is burning or gnawing, more or less continuous, but intensified by movement or manipulation. In severe cases, other disturbances of nerve-function occur. These are tingling, formication, and anæsthesia, in connection with sensory fibres; and atrophy of muscles, muscular weakness, and sometimes fibrillary tremors, from implication of motor fibres. The electrical reactions are not markedly altered except in severe cases, when reaction of degeneration may occur.

Diagnosis.—The diagnostic points in favour of sciatic neuritis are the tenderness of the nerve and the presence of anæsthesia and muscular atrophy. In these it differs from a pure neuralgia of this region. Further it must be distinguished from the pains due to *hip-joint disease, sacro-iliac disease, pelvic lesions,* and *tumours of the*

femur, in which, also, tenderness of the nerve should be absent, and the pains are more limited to the seat of the lesion. But even if neuritis is present, it may be secondary to such lesions, and the symptoms special to them should be carefully looked for before concluding that the disease is a primary sciatica.

Prognosis.—This is, on the whole, favourable, but the duration is very variable. Slight cases may recover quickly; severer cases last months or years; and after subsidence of the pain, the muscular wasting, fibrillary contractions, and a tendency to cramps may persist for some time.

Treatment.—Complete rest is essential, in the position that most eases the pain. If there is good evidence that gout is an antecedent, suitable remedies, colchicum, lithium salts, and saline aperients should be given, and the diet modified by reduction of its nitrogenous elements. In other cases sodium salicylate, aspirin in full doses, and potassium iodide are of use. In acute cases—especially when exposure to cold seems to have been the cause—hot poultices or fomentations should be applied to the affected limb. In later stages, both counter-irritants and sedatives are of value, and massage may be tried. Mustard plasters, blisters frequently repeated, acetic turpentine liniments, acupuncture along the course of the nerve, and the chloride of methyl spray as recommended by Debove, may all give some relief. This last is only a method of obtaining counter-irritation by temporary freezing of the skin along the course of the nerve. Often the greatest benefit is obtained from injections of cocaine ($\frac{1}{2}$ to 1 grain), and of morphia; but they must be given with a full recognition of the fact that a serious "habit" or "craving" may be induced. Belladonna, opium, and chloroform liniments are useful anodynes; and the daily or more frequent injection of ·5 c.c. of normal saline solution under the skin at the most painful spots is said to be efficacious. Electricity may be used as a constant galvanic current of from 30 to 50 milliampères, with large electrodes, the positive applied to the iliac region, and the negative moved up and down the back of the thigh and the calf for fifteen or twenty minutes. In later stages the electric dipolar bath with sinusoidal currents may be tried (Lewis Jones). Finally, in severe cases, nerve-stretching remains as a means of getting relief, generally for some time, even if the pain subsequently recurs; in very old cases, where only movement gives rise to pains, which may then be attributable to adhesions, massage and manipulations may do good.

MERALGIA PARÆSTHETICA

This is an affection, probably inflammatory, of the external cutaneous nerve of the lumbar plexus. The symptoms are *pain*, sometimes brought on by walking, at others when the patient is lying still or sitting, and abnormal sensations, such as numbness, pins and needles, cold feeling, burning, sensation of tightness, felt on the front and outer part of the thigh within the distribution of

the above nerve. Over the same area there is some modification of sensibility, either hyperæsthesia, or more often anæsthesia, or changes in the appreciation of pain, heat or cold. In most cases there is some tenderness on pressure below the anterior superior iliac spine, where the nerve comes through the fascia lata. It is more frequent in men than in women, and has been attributed in different cases to injury and to toxic and infective agents. Rest, warm baths, and the continuous galvanic current should be tried for its treatment.

LOCALISED MUSCULAR SPASM

A number of local disorders are recognised, in which clonic or tonic spasm of muscles supplied by a particular nerve is the main feature. They are sometimes functional, at others dependent on organic disease. Some of them are described in other parts of the volume, namely, *conjugate deviation* of the eyes and head (*see* Hemiplegia), laryngeal spasm, and spasmodic wryneck.

SPASM OF OCULAR MUSCLES

This occurs in association with various diseases of the eye, in the conjugate deviation of cerebral disease, in hysteria, and other conditions. Clonic spasm is termed *nystagmus*. The eyes are moved rapidly to and fro, generally in a lateral direction, sometimes vertically. It results from some ocular defect, such as extreme choroidal atrophy and albinism. It occurs in a number of central nervous diseases, with greatest constancy in disseminated sclerosis and Friedreich's ataxy, but also frequently in tumours of the cerebellum. It also appears in miners, especially in those who work in the recumbent position.

SPASM OF THE JAW

Trismus, or spasm of the muscle closing the jaw, is one of the first indications of the onset of tetanus. The jaw is fixed by tonic contraction of the masseter or temporal muscles, so that the teeth cannot be separated more than a few lines. A similar spasm may be due to irritation of the teeth, or to stomatitis, or, on the other hand, to central disease, such as disease of the pons in the neighbourhood of the fifth nerve-nucleus. It must be distinguished from tumours or rheumatic arthritis fixing the jaw-joint. *Clonic* spasm of the jaw occurs in rigor, in convulsions, and in hysteria—rarely as an isolated phenomenon.

FACIAL SPASM

Irregular contractions of the facial muscles take place in chorea, and a tonic contraction is a late stage of facial paralysis. Boys and girls often acquire a habit of twitching certain muscles of the face,

neck, or other part of the body, and this habit may last into adult life (*see* Habit Spasm).

More serious cases of facial spasm (*convulsive tic, histrionic spasm*) occur in people over twenty years of age, and mostly between thirty and sixty. In some of these there is actual irritation of the facial nerve by tumours in the pons, or of the facial cortical centre on the opposite side of the brain; in most cases the condition seems to be functional. It is much more frequent in women than in men, and arises from emotion, mental anxiety, irritation of the peripheral branches of the fifth nerve as in the eyelids, or the teeth, and from exposure to cold. The spasm chiefly affects the orbicularis palpebarum (*blepharospasm*) and the zygomatici, so that the eye is half closed, and the angle of the mouth is drawn up. Other facial muscles, including the platysma myoides, are also contracted, but the orbicularis oris and frontalis muscle, as a rule, escape. The contractions are momentary, and frequently repeated; or the spasm is of longer duration, and recurs at longer intervals; but it causes no pain. The spasm is at first entirely on one side, and only in severe or prolonged cases affects the other side. The electrical reactions are usually normal. (*See* General Convulsive Tic.)

The **Prognosis** in a well-established case is unsatisfactory; the disease will last months, or years, and even to the end of life.

The **Treatment** consists in the removal of causes of irritation, if they can be recognised; the use of nervine tonics, such as zinc sulphate, iron perchloride or sulphate, and strychnine; and of sedatives, especially the hypodermic injection of morphia. A weak galvanic current applied continuously, and counter-irritation by blisters behind the ear, may be tried. Nerve-stretching has been performed in some cases, but only exceptionally with any lasting benefit.

HICCOUGH

This is a repeated convulsive contraction of the diaphragm, induced reflexly, as a rule, by irritation in the abdominal cavity; as by a full meal, pepper or spices taken into the stomach, or by peritonitis. It is also the result sometimes of excessive laughing. Persistent hiccough is due to peritonitis or other abdominal lesions, or is a part of hysteria. Hiccough may be stopped by holding the breath, by taking spiritus ætheris nitrosi, or other diffusible stimulant: in protracted cases inhalations of chloroform, or nitrite of amyl, or injections of morphia may be necessary. Sometimes firm traction on the tongue is successful.

DISEASES OF THE SPINAL CORD

PRELIMINARY CONSIDERATIONS

It will not be necessary to go into full detail as to the anatomy of the spinal cord. The familiar appearance of gray cornua and white columns as seen on transverse section is shown in the accompanying figure, and the further subdivisions of the white columns are there indicated.

The *pyramidal tract* descends from the cortex of the brain through the internal capsule, crus cerebri, pons varolii, and medulla oblongata to reach the spinal cord. In the medulla oblongata the greater part of it *decussates* to the opposite side, the *crossed pyramidal tract;* it occupies the posterior half of the lateral column, outside and in front of the posterior cornu. In the lumbar region it reaches the surface of the cord, but higher up it lies within the cerebellar tract. Its fibres, which are the axons of the upper *motor* neurons, pass into the anterior cornua, where its terminal arborisations surround the cell-bodies (motor nerve-cells) of the lower motor neurons.

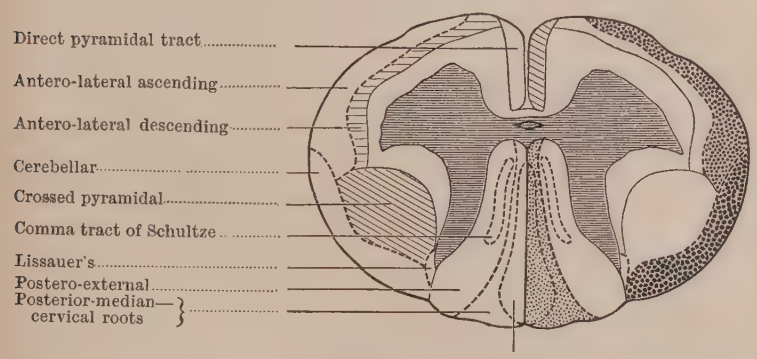
The smaller portion of the pyramidal tract, which does not decussate in the medulla oblongata, is called the *direct* pyramidal tract, or column of Türck. It lies on the inner side of the anterior column; and as it passes down, its fibres gradually cross the middle line in the anterior commissure to reach the opposite anterior cornua, so that in the lumbar region it is no longer visible.

The posterior columns (*postero-external,* or column of Burdach, and *posterior median,* or column of Goll) contain chiefly the axis-cylinder processes of the lower sensory neurons, on the same side as their cell-bodies. The *antero-lateral ascending* tract (Gowers) begins above the lumbar cord; it contains the axons of middle (or upper) sensory neurons, the cell-bodies of which lie in the posterior gray cornua on the opposite side of the cord: they are therefore decussating tracts. The *cerebellar tract* begins in the middle of the lumbar region; it contains the axons of sensory neurons, of which the cell-bodies form Clarke's columns; and the fibres terminate in the cerebellum. It is known further that impulses are transmitted downwards by the *antero-lateral descending tract* and by the *comma tract.*

Effects of Lesions.—In relation to disease the cord is, first, a means of transmitting impulses between the brain and the limbs and other parts of the body, and this is effected by the system of neurons already described (*see also* p. 196). Secondly, the cord is

a centre for reflex action, by means of the cell-bodies of the lower motor neurons contained in the anterior cornua. Thirdly, these nerve-cells at the same time control the nutrition of the muscles,

FIG. 25



Transverse Section of Spinal Cord, showing Tracts of White Matter.
(*After Sherrington.*)

while the nutrition of the skin and other parts seems to be related to the sensory neurons.

If the *anterior gray cornu*, at any level, be alone affected by any lesion, the immediate result is paralysis of the muscles in connection with it, and loss of reflexes in the corresponding area. If the lesion is severe or protracted, degenerative effects take place in the nerve, of the same kind as those which follow neuritis and lesions of the nerves, and as a consquence atrophy of nerves, atrophy of muscles (*amyotrophy*), altered electrical reactions, and nutritive changes are observed.

If a lesion is confined to the *white column*, there will be loss of conduction of motor or sensory impulses, according to the situation of the lesion—that is to say, there will be paralysis or anæsthesia, or other form of sensory disturbance, or inco-ordination of movement, or some combination of these. But so long as the gray centres (and anterior root fibres) are untouched there will be no pronounced atrophy and no degenerative reaction.

But the white columns of the cord consist of nerve-fibres (axons), and just as the nerve-trunks themselves degenerate when their fibres are cut off from their centres, so in the cord a *secondary degeneration* takes place when certain tracts are affected so as to interrupt their connection with their functional centres. This degeneration takes place in the direction of conduction of impulses—that is, downwards (or peripherally) in the case of motor neurons (*descending degeneration*); and upwards (or centripetally) in the case of sensory neurons (*ascending degeneration*). Thus, a lesion in the centre of

the cord destroying or severely compressing the white columns causes degeneration of the pyramidal tract, direct and crossed, in the part of the cord below the lesion, and sometimes of the small comma tract of Schultze; and of the posterior median column, cerebellar tract, and antero-lateral ascending tract in the part of the cord above. The postero-external column also degenerates for a short distance above the lesion; but the cerebellar tract is unaffected by lesions below the junction of the lumbar and dorsal portions. As will be shown later, unilateral cerebral lesions involving the pyramidal tracts also cause a descending degeneration of the pyramidal tract, which affects the direct tract on the same side, and the crossed tract on the opposite side of the cord; obviously in such a case the sensory columns are not affected. To the naked eye the areas of secondary degeneration are of a reddish or yellowish-gray colour, but in early stages they may be scarcely visible. They may then be demonstrated by various staining reagents.

The process of degeneration is similar to what takes place in the nerves: destruction of myelin, disappearance of axis-cylinders, increase of connective tissue (or neuroglia), and later the formation of granule-corpuscles—large cells filled entirely with small granules of fat, as a result of which they have a dark colour on ordinary examination, but disappear in the usual processes for clearing sections. In long-standing cases the connective tissue develops, the parts become hard and dense, and the name *sclerosis*, which is scarcely suitable in earlier stages, may be properly used.

Certain functional changes result from lesions of the pyramidal tract. These are: (1) Rigidity of the muscles corresponding to the part of the spinal cord below the lesion; (2) increased reflex irritability. They have been attributed to the secondary degeneration, and do, indeed, show themselves, not immediately, but after an interval which corresponds closely with the time required for the occurrence of the change in the nerve. But the sclerosis acts, probably, not by irritating the anterior cornua, but by the removal of the inhibitory control of the higher centres, so that the lower centres are unduly excitable.

The increased reflexes take place in the part of the spinal cord which is itself healthy. The knee-jerk is excessive; foot-clonus is easily produced; Babinski's sign is observed; and sometimes the knee-jerk is followed by a temporary clonus. Pinching the skin causes retraction of the limb after a definite interval.

No symptoms have hitherto been referred to the *ascending degeneration* which follows a lesion affecting the sensory tracts.

From the above it will be seen that, with respect to motor disturbances, the results depend upon the relation of the lesion to the upper and lower neurons.

If the lower neurons contained in the anterior cornua and anterior roots are injured, the condition is comparable with that which follows lesion of a motor nerve (*see* Neuritis). Voluntary motion, spinal reflexes, nutrition and electrical reactions are all

dependent upon the integrity of the gray cornua, or the motor fibres. Hence lesions of these parts destroy these functions, and, as a result, there occur paralysis, loss of reflexes, degeneration of nerves, atrophy of muscles, and reaction of degeneration.

If the upper neurons contained in the pyramidal tracts are alone injured, voluntary motion is indeed lost, because the conducting fibres from the brain to the spinal centres are interrupted; but the functions which depend solely on the integrity of the gray matter, nerve roots, and fibres are maintained—namely, the nutrition of nerves and muscles and their electrical reactions. The spinal reflexes are in most cases increased.

Primary Disease of the Neurons.—The several groups of neurons in the spinal cord may be separately affected by degeneration or disease. Such degeneration is the result of toxins, or is due to congenital want of vitality, or remains entirely unaccounted for. The lesions have been known as *tract diseases* or *system diseases*, and the following are instances: Tabes dorsalis, in which the lower afferent or sensory neurons are diseased: Spastic paraplegia—upper motor or efferent neurons: Ataxic paraplegia and Friedreich's disease—lower afferent and upper efferent neurons: Progressive muscular atrophy—lower motor neurons: Amyotrophic lateral sclerosis—upper and lower motor neurons.

Transverse Lesions.—The spinal cord, having an elongated form, is naturally liable to lesions affecting its whole thickness, such as may occur from external pressure, the indiscriminate growth of tumour, the diffuse spread of inflammation, or the anæmia of vascular obstruction. In such cases gray and white matters are equally affected. The results of such a transverse lesion show themselves mainly as an interruption of the *conducting* power of the cord; but if the lesion is at all extensive vertically, its effects upon the *nerve-centres* must also be considered—that is, both upper and lower neurons are affected. The results also vary, according as the lesion is *bilateral* or *unilateral*, in consequence of differences between the motor and sensory fibres in the process of decussation.

RESULTS OF A BILATERAL TRANSVERSE LESION AS AFFECTING CONDUCTION

Paralysis of all muscles below the lesion.

Anæsthesia of parts below.

Functions of bladder and rectum impaired. Trophic relations and electrical reactions continue normal.

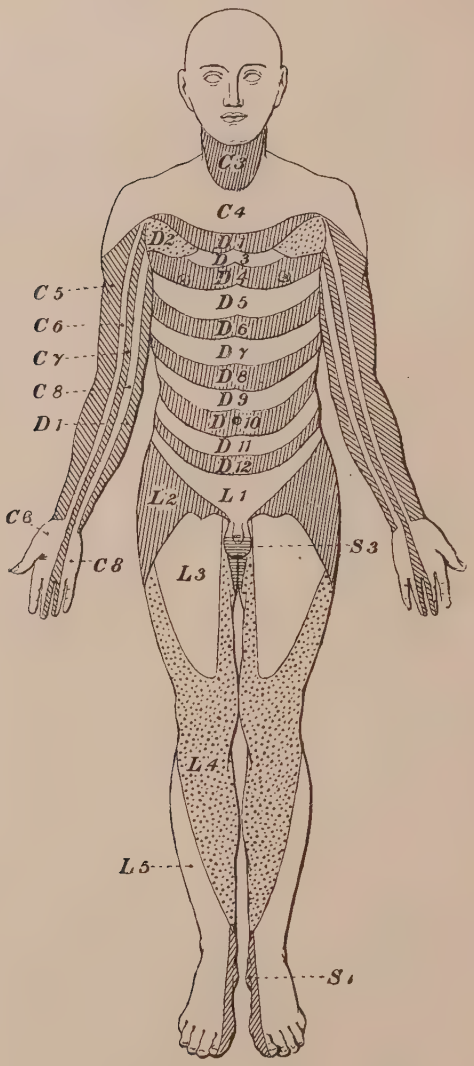
Subsequently, muscular rigidity and increased reflexes.

This last statement requires modification, for when a transverse lesion of the spinal cord is *complete*, so as to interfere absolutely with all impulses passing through the lesion, the deep reflexes are not increased, but are abolished, and the muscles are flaccid (Bastian and others). Nevertheless, in most cases of transverse

Segment.	Muscles.	Reflex and Centres.	Sensation.
First Cervical.	Rectus lateralis. Rectus capitis anticus and posticus. Sterno-hyoid. Sterno-thyroid.		
Second and Third Cervical.	Sterno-mastoid. Trapezius. Scaleni and neck. Omo-hyoid. Diaphragm.	Hypochondrium (?) Sudden inspiration produced by sudden pressure beneath the lower border of the ribs.	Back of head to vertex. Neck.
Fourth Cervical.	Diaphragm. Deltoid. Biceps. Coraco-brachialis. Supinator longus. Rhomboid. Supra- and infra-spinatus.	Pupillary (fourth cervical to second dorsal). Dilatation of the pupil produced by irritation of the neck.	Neck. Shoulder, upper surface. Outer surface of arm. Anterior thorax as far as second rib.
Fifth Cervical.	Deltoid. Biceps. Coraco-brachialis. Brachialis anticus. Supinator longus. Supinator brevis. Rhomboid. Teres minor. Pectoralis (clavicular part). Serratus magnus.	Scapular (fifth cervical to first dorsal). Tendon reflexes of corresponding muscles.	Back of shoulder and arm. Outer side of arm and forearm to the wrist.
Sixth Cervical.	Biceps. Brachialis anticus. Extensors of hand and fingers. Pectoralis (clavicular part). Serratus magnus. Triceps. Pronators.	Tendon reflexes of extensors of the arm and forearm. Posterior wrist (sixth to eighth cervical). Tapping tendon causes extension of hand.	Back and front of arm and forearm within above. Radial side of hand to middle of second finger.
Seventh Cervical.	Triceps (long head). Extensors of hands and fingers. Pronators of hand. Flexors of hand. Subscapular. Pectoralis (costal part). Latissimus dorsi. Teres major.	Palmar (seventh cervical to first dorsal). Stroking palm causes closure of fingers.	Back and front of arm and forearm within above, and hand to middle of third finger.

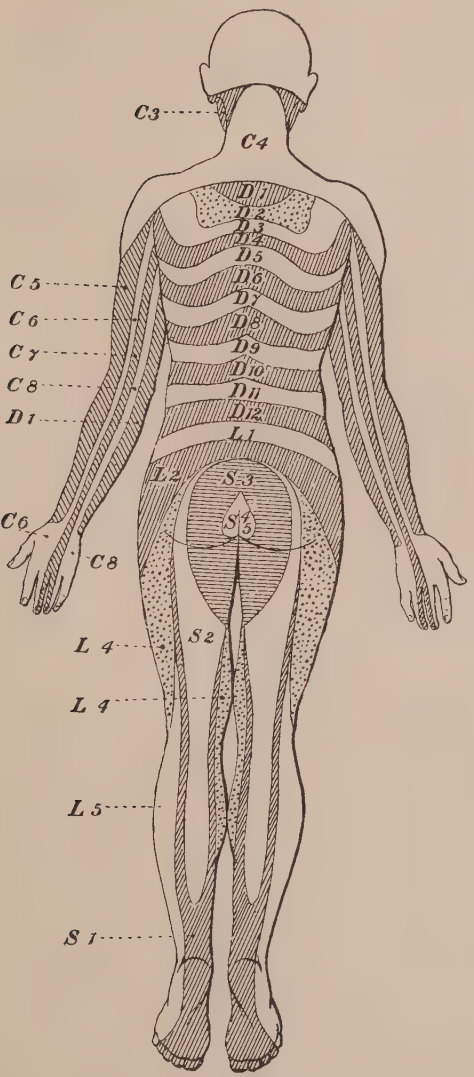
Segment.	Muscles.	Reflex and Centres.	Sensation.
Eighth Cervical.	Flexors of hand and fingers. Intrinsic hand muscles.	—	Back and front of arm and forearm within above. Ulnar area of hands, back and palm.
First Dorsal.	Extensors of thumb. Intrinsic hand muscles. Thenar and hypothenar muscles.	—	Inner side of arm and forearm to wrist.
Second to Twelfth Dorsal.	Muscles of back and abdomen. Erectores spinæ.	Epigastric (fourth to seventh dorsal). Abdominal (seventh to eleventh dorsal). Vasomotor centres (second dorsal to second lumbar).	Skin of chest, back, and abdomen, in bands running around and downwards, corresponding to spinal nerves. Upper gluteal region.
First Lumbar.	Ilio-psoas. Sartorius. Abdominal muscles.	Cremasteric (first to third lumbar).	Skin over groin and front of scrotum.
Second Lumbar.	Ilio-psoas. Sartorius. Flexors of the leg (Remak). Quadriceps femoris.	Patellar tendon (second to fourth lumbar).	Outer side and upper front of thigh.
Third Lumbar.	Adductors and inward rotators of thigh. Flexors of thigh.	—	Front and inner side of thigh.
Fourth Lumbar.	Adductors of thigh. Abductors of thigh. Tibialis anticus. Flexors of leg (Ferrier).	Gluteal (fourth to fifth lumbar).	Inner side of thigh and leg to ankle.
Fifth Lumbar.	Outward rotators. Flexors of leg (Ferrier). Flexors of foot. Peronei. Extensors of toes.	—	Outer side of leg, dorsum of foot, and outer part of sole.
First and Second Sacral.	Flexors of foot and toes. Peronei. Small muscles of foot.	Plantar (fifth lumbar to second sacral).	Back of buttock, thigh, and leg, inner side of foot and sole.
Third to Fifth Sacral.	Muscles of the perineum.	Tendo Achillis. Vesical centre. Anal centre. Sexual centre.	Sacral region, anus, perineum, and genitals.

FIG. 26



The figure represents some of the opinions which have been expressed as to the areas of skin corresponding to the sensory roots of the Spinal Nerves. It may be compared with the fourth column on pp. 258–259 (*see Brain*, 1893, 1894 ; *Amer. Journ. of Med. Sci.* 1888, 1892).

FIG. 27



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myelitis, perhaps because the lesion is incomplete, the reflexes are increased and the muscles are rigid.

RESULTS OF A UNILATERAL TRANSVERSE LESION AS AFFECTING CONDUCTION

On the same side as the lesion.	On the opposite side.
Paralysis of muscles below.	Anæsthesia of skin below.
Hyperæsthesia of skin below.	Normal muscular power, muscular sense, reflex action and temperature.
Diminished muscular sense and sensibility.	
Reflexes at first lessened, then increased.	
Vasomotor paralysis and elevation of temperature.	
Nutrition and electrical reactions retained.	

To these symptoms in each case may be added those which are due to the changes at the level of the disease.

In a total transverse lesion these are: paralysis, atrophy, reaction of degeneration, and loss of reflexes in the muscles supplied by nerves directly connected with the injured segment; anæsthesia, with a band of hyperæsthesia at the same level.

In a one-sided lesion, the symptoms just enumerated occur on the side of the lesion; on the opposite side, anæsthesia alone is found at the same level.

In the accompanying Tables and Figures (pp. 258–261), compiled from the work of Allen Starr, Thorburn, Head, and others, the relations of the segments of the spinal cord, as represented by nerve-roots, to the muscles, the reflexes, and the cutaneous sensory areas, are indicated.

The segments, however, are not independent of one another. Every single movement involves more than one muscle and more than one spinal segment. Stimulation of a spinal root only moves a part of a muscle, and section of it only paralyses a part of a muscle. At least three segments must be damaged to paralyse a muscle completely. One and the same segment contains fibres for a muscle and for its antagonist.

The skin areas shown on the limbs in Figs. 26 and 27 have a generally longitudinal arrangement; but cases of limited anæsthesia of the limbs occur in which the anæsthetic areas have transverse limits, that is, limits perpendicular to the axis of the limb, corresponding for instance to the space covered by a glove or a sock. Such a transverse distribution has been attributed to a lesion of the *middle* neurons.

ACUTE MYELITIS AND SOFTENING OF THE SPINAL CORD

The very general adoption of the view that symptoms and pathological changes hitherto attributed to myelitis are really in many cases the result of softening from vascular occlusion (thrombosis or embolism) makes it desirable to deal with these two conditions together.

Inflammation of the spinal cord, or myelitis, occurs in several forms. As an acute disease, it may be a *general* or *disseminated*, affecting white and gray matter indiscriminately throughout the cord; it is sometimes *localised* or *focal*. It is, in a certain class of cases, confined to the anterior cornua of gray matter, and is then known as *poliomyelitis*, one cause of acute or atrophic spinal paralysis (infantile and adult): this will be separately described.

Softening of the cord (*myelomalacia*) is probably the cause of the greater number of cases in which a sudden limited lesion occurs in the spinal cord, causing paraplegia, and of the chronic cases arising out of these conditions.

Ætiology.—True acute myelitis may be the result of specific *infections*, such as those of enteric fever, small-pox, diphtheria, influenza, gonorrhœa, and especially syphilis. In some cases apparently related to cold and wet, micro-organisms (streptococci and diplococci) have been found in the diseased parts. But, as a rule, the organisms cannot be detected, and even after the experimental production of myelitis by the introduction of bacteria into the cerebro-spinal fluid, or blood, the cord may show no organisms; so that the changes must be attributed to toxins. Myelitis appears sometimes to arise from injuries and strains of the back, such as those due to lifting great weights. It is set up by *adjacent inflammations*, such as meningitis; it is sometimes caused, with meningitis, by bed-sores ulcerating into the spinal canal; and a very frequent local cause is *compression* of the spinal cord by tumours, caries, and inflammatory growth in the membranes.

Softening of the cord is chiefly due to thrombosis following upon arterial disease: and this is very frequently syphilitic arteritis, and in other cases the arterio-sclerosis of advancing years. No doubt, also, sprains and shocks, as well as infective toxins, may induce thrombosis in small spinal vessels apart from true inflammatory change.

Anatomical Changes.—The cord in myelitis is softened, and bulges on transverse section, or may be quite diffluent; but previous to section it may seem hard, from mere tenseness of the membrane containing the swollen cord.* To the naked eye the section is congested, minute vessels are visible, especially in the gray matter, and this itself is darker than usual, and its outline is indistinct. Patches of gray tint may be present in the white column, or the

* Any cord may become soft from decomposition, or may be reduced to a pulp by clumsiness in extracting it from the spinal column.

whole surface is confused. Under the microscope, in early stages, are found perhaps capillary hæmorrhages; and leucocytes in the lymphatic sheaths and around the vessels. The nerve-fibres suffer by breaking up of their myelin, and here and there occurs a fusiform enlargement of the axis-cylinders, which in its thickest part is five or six times the diameter of the normal fibre: this may be due to imbibition of fluid. The nerve-cells are swollen, granular, with perhaps fatty globules, and some of them undergo vacuolation. Subsequently both nerve-cells and fibres degenerate, the connective tissue increases in quantity, the cells, known as Deiters' cells, are larger and more numerous, and granule-cells are formed in abundance. Finally, in cases of long standing, or passing into a chronic condition, the granule-cells disappear also, and the new connective tissue becomes firm, dense, even finely fibrous, so that a definite *sclerosis* is the result. An actual *abscess* of the spinal cord is rare.

In cases of pure softening the disintegration of the nervous tissues is very similar, but the vessels show degenerative changes, and, at any rate at first, leucocytal infiltration is confined to the neighbourhood of the vessels. Ultimately, however, sclerotic changes occur in the neuroglia as in the purely inflammatory cases.

The following differences in the seat of the lesion are distinguished: it is called *focal* or *localised* when very limited in extent; *diffuse* when it is of considerable extent continuously; *disseminated* when there are numerous scattered patches over a large extent; *transverse* when the lesion extends across the cord and is of little vertical extent. *Central* and *peripheral* or *annular myelitis* are also spoken of.

A localised or transverse lesion persisting for a certain time is followed by ascending and descending secondary degenerations (*see* p. 255).

Symptoms.—Myelitis mostly begins with numbness or tingling in the extremities, and in acute cases there may be malaise or slight febrile reaction, and perhaps rigors; or there are painful sensations in the extremities, or hyperæsthesia, or pains in the back or muscles. Twitchings and tremors of the muscles, or cramps and spasms, may also occur as early symptoms, indicating a stage of irritation. Within a day or two, or even in a few hours in the most acute cases, paralysis and anæsthesia become well marked.

As a general rule, the symptoms in thrombotic softening are much more sudden in their onset, paralysis and anæsthesia occurring with little or no warning; and with no pain, or certainly less than is usually the case in hæmorrhage.

The symptoms in detail vary according to the nature of the change and the extent of cord involved.

Acute diffuse and *disseminated myelitis* are comparatively rare. Numbness and tingling, weakness, and then paralysis rapidly develop. The patient is more or less completely paralysed in all four limbs, and in the abdominal and intercostal muscles; there are general anæsthesia, loss of reflexes, retention of urine, and involuntary passage of fæces. The temperature rises to 102° or 103°; the

breathing becomes seriously involved, and death often takes place in from three days to three or four weeks. It is the disseminated form which is more likely to occur after specific fevers. It is occasionally accompanied by optic neuritis.

A form of disseminated myelitis, combined with a similar lesion of the brain, is described under the name of *acute disseminated encephalo-myelitis* (*acute central ataxia* of Leyden); numerous small foci of inflammation are found in the cord, the pons, and the cerebrum. It nearly always occurs during or soon after an infectious disease or similar process; consequently a large proportion of the cases are in children. The onset is sudden, and suggests the implication of the whole cerebral nervous system. There is often unconsciousness, either with paralysis, or with restlessness and delusion; and after a time the unconsciousness clears up, and a stage is reached which is characterised by ataxia or choreic movements. In other cases the sudden cerebral symptoms are absent, and ataxia is the first symptom. In this stage there are ataxic movements or intention-tremor of the legs, arms, and head; disorders of speech, especially of the scanning variety, but sometimes dysarthric; and sometimes disturbances of intellect. The gait is variable; it may be ataxic, or spastic, or paretic, or tremulous. The arms are less commonly ataxic, but more often present intention-tremor; and the same occurs with the head. There is great variety in the subsequent course: a few cases recover quickly and permanently; many recover after months, or after one or two relapses; in other cases, some symptoms, especially speech disturbances, persist; but death is rare. Aphasia or optic neuritis occasionally occurs in the earliest stage. The resemblance in many points to disseminated sclerosis (*see* p. 298) is obvious, but nystagmus appears to be uncommon.

Localised and *transverse* lesions are more generally determined by softening. They are often situate in the *mid-dorsal* or *lower dorsal* region. The characteristic symptoms then are paralysis and anæsthesia of the lower extremities, constituting the ordinary form of *paraplegia*. In severe cases the patient lies in bed, unable to raise the leg from the bed, or even move a toe. There is more or less complete numbness, and loss of sensation in the legs, thighs, and lower part of the abdomen, up to a transverse line about the level of the umbilicus, higher or lower according to the exact position of the lesion. This upper limit of anæsthesia is generally sharply defined, and at the same level there is often a narrow band of *hyperæsthesia;* and a painful sensation known as *girdle-pain* extends from the seat of the lesion round each side of the body to the front. The reflexes are commonly increased. Constipation is usually present; the bladder may be at first paralysed, and retention occurs. After a short time, since the lesion is above the spinal centres for the bladder, the power of expulsion is restored, but from interruption of the communication between the bladder and the brain the sensation of distension is not experienced, and

micturition is performed unconsciously and uncontrolled. Bed-sores readily form over the sacrum, over the trochanters, and on the heels, partly as a result of trophic disturbance, partly as a result of the continuous local pressure which follows when the patient can neither feel the pain of pressure nor shift the limb to escape it. Coincidently with the occurrence of secondary degenerations, the deep reflexes are increased; knee-jerk is exaggerated; ankle-clonus and Babinski's sign are observed; and pinching the skin of the dorsum of the foot causes drawing up of the leg, which the patient himself can by no effort move. Later on, the muscles begin to get rigid, either as a permanent condition, or as a temporary reflex result of moving or handling the limb. Thus, by placing the hand under the knee to lift it, the limb may at first be felt to be flaccid, but quickly stiffens, and remains rigid for some time. The excitability of the lower part of the cord may be so great that the slightest touch or shock to the bed, or the passage of a catheter, or the discharge of urine, causes the paralysed leg to be flexed forcibly upon the abdomen. The electrical condition of the muscles differs but little from the normal; they respond to both galvanic and faradic currents—perhaps somewhat less than in health. If the transverse lesion is *complete*, so as to divide all the nerve-tracts in the cord, the muscles become flaccid and the reflexes are lost, even though there is no extension of the lesion downwards to the gray matter which forms part of the reflex paths.

When the *lumbar portion* of the cord is the seat of disease, it is again the legs that are affected with anæsthesia and paralysis; but now it is not only that their communications are cut, but that their motor centres are destroyed. The muscles undergo atrophy, and give the reaction of degeneration when tested electrically. The reflexes, which require the integrity of the lumbar portion of the cord as part of the reflex arc (*see* p. 207), are lost. The lowest part of the lumbar cord also contains the centres for the rectum and the bladder; and, if this is involved, the sphincters are paralysed, and there is incontinence of urine and fæces.

When the lesion occurs in the *cervical region* of the cord, the condition of the lower part of the body is practically the same as that which results from a dorsal lesion. But other parts are involved, which make this a much more serious form of disease. The paralysis extends to the arms, and from implication of the corresponding gray matter there may be loss of reflexes in the arms, while those of the lower extremities are increased. The intercostal muscles are paralysed, and if the lesion is sufficiently high the diaphragm is also involved (*see* p. 243). This readily leads to pulmonary complications: the lungs become congested and œdematous, the bronchial tubes are filled with mucus, and the patient may die asphyxiated. The heart may beat rapidly or irregularly. Sometimes, as in some other affections involving the cervical cord (tetanus, meningitis, fractured spine), *hyperpyrexia* occurs, the temperature rising to 107°, 108°, or 110° F.; and *priapism* is occasionally observed.

Other symptoms of cervical lesions are contraction of the pupil, and diminution of the palpebral fissure, dysphagia, hiccough, and very slow or very quick pulse.

Variations of the symptoms may also occur as a result of the lesion being limited to one side, or to one small portion of the transverse section; or two or more patches of softening may occur in different parts of the cord.

Death in cases of acute myelitis or softening takes place (1) from pulmonary complications following upon paralysis of the respiratory muscles; (2) from bed-sores intensifying exhaustion, or leading to pyæmia; (3) from vesical complications; (4) from intercurrent affections, such as pneumonia or bronchitis. The bladder is extremely liable to cystitis, partly from trophic disturbance, partly from retention of urine. Sometimes the use of a catheter is responsible for the introduction of organisms which may set up septic inflammation. When cystitis occurs the urine rapidly decomposes and becomes ammoniacal, unless it is repeatedly removed by the catheter; it contains pus, or muco-pus, and readily deposits crystals of ammonio-magnesian phosphate on standing. Almost at any time the septic condition of the bladder may extend up the ureters to the kidneys, and suppurative pyelitis and nephritis will then occur, and the patient may die with uræmic symptoms. Sometimes death occurs from the condition which has been described as "catheter fever" and "urethral fever."

Cases which escape these dangers generally lapse into a chronic condition, which may be of indefinite duration, but sometimes recovery slowly takes place after many months. And a small number of cases get well comparatively quickly, and these are more common among the milder cases of myelitis following infectious disorders. Myelitis from compression in caries of the spine often gets better; acute diffuse myelitis is mostly fatal.

Diagnosis.—Myelitis and myelomalacia may be confounded with spinal meningitis, hæmorrhage into the spinal cord or membranes, multiple neuritis, and hysterical conditions.

In *spinal meningitis* there is usually more evidence of irritation of the nerve-roots, shown by radiating and local pains, hyperæsthesia, and muscular spasm; and febrile reaction is more constant and persistent. *Hæmorrhage* into the spinal cord is generally quite sudden, severe local pain being quickly followed by paralysis below the lesion, whereas in myelitis, as a rule, the paralysis develops more gradually, with some preceding symptoms of sensory or motor irritation, and possibly febrile reaction for a few days in myelitis. In softening the pain is less than in hæmorrhage. In *meningeal hæmorrhage* the onset is also sudden, and the signs of irritation of the nerve-roots are more pronounced than in lesions of the cord-substance. Cases of *multiple neuritis* have no doubt been frequently regarded as instances of myelitis. The points in favour of neuritis are the affection of all four limbs simultaneously, the early predominance of extensor paralysis of the hands and feet, and the implication of the face and larynx in some cases; the co-existence

in the parts affected of anæsthesia with muscular atrophy, altered electrical reactions, and diminished reflexes, showing a universal interference with the sensory and motor paths at or below the trophic centres (*i.e.*, of lower neurons), such as can only occur from lesions of many nerve-trunks, or from a diffuse lesion of the cord throughout its whole length. In neuritis there is also tenderness of nerve-trunks in exposed situations, and of the muscles, especially those of the calf. A history of alcoholism, or the mental condition previously described (*see* p. 218), is in favour of neuritis, and a very gradual onset is perhaps more frequent in neuritis than in myelitis.

From *hysterical paralysis* the diagnosis is often most difficult; the history of the patient previous to hysterical attacks, or the first appearance of the symptoms after some emotional disturbance, may justify a suspicion of hysteria; but the case must be carefully examined from the point of view of structural lesions, since hysterical people are not excluded from the possibility of organic disease. Patients with hysterical paraplegia do not develop the rigidity and increased reflexes of transverse lesions of the cord, nor do they get atrophy of the muscles, bed-sores, incontinence of urine and fæces, or cystitis; nor do they have girdle-pain. It is especially slight cases that are apt to be set down as hysteria; where the patient can perhaps stand, but her inability to put one leg before the other is attributed to unwillingness. A careful examination of the muscular power while sitting or in bed, frequently repeated to test its constancy, and the use of electrical tests, will probably guard against this. It must be allowed that a good deal of weakness of the lower extremities may arise in persons, who are not hysterical, from purely *functional* causes, such as exhaustion from long illnesses, or prolonged over-exertion, when it may be supposed that the functions of the cord itself are at fault rather than those of the brain, as in pure hysteria.

When other diseases have been excluded it yet remains to form an opinion as to the position of the lesion, which in a transverse myelitis may generally be determined by a consideration of the symptoms in relation to the Table and Figures (*see* pp. 258–261). It is important always to think of caries of the spine, and one should ask for a history of blow or strain, and look for any undue prominence of a vertebral spine; and this frequently, for paraplegic symptoms may precede by several months the appearance of angular curvature or kyphosis.

Treatment.—Rest is, of course, essential, and in severe cases, or cases likely to be of long duration, a water-bed should be provided to avoid the risk of bed-sores. It is very doubtful if any local treatment can be of value; but in the past hot fomentations, hot-water bags, mustard-plasters, stimulating liniments, ice-bags, leeches and dry-cupping have at different times been applied to the spine.

Internally, potassium iodide and mercuric chloride are frequently given, but without much evidence that they have any effect in lessening the inflammation. In cases due to syphilis, a course of mercury should certainly be instituted, and continued or repeated

over a long period. Ergot, or ergotin injected subcutaneously, is supposed to diminish vascular engorgement, and may perhaps be most useful if hæmorrhage has any share in the lesion. Sodium salicylate may very properly be tried in cases apparently owning a toxic or febrile origin. The greatest care must be taken to prevent bed-sores by relieving the pressure on prominent parts, by keeping the skin perfectly clean, washing it daily with spirit lotion, dusting the sheet beneath it with oxide of zinc or starch powder, and changing this whenever it becomes moist from any cause. The bladder, also, must be constantly attended to. If the urine is retained, it must be drawn off with the catheter two or three times daily, with antiseptic precautions. If cystitis occurs, and the urine becomes alkaline and offensive, antiseptic injections may be used; and urotropin or salicylic acid should be given internally.

The diet should be light and nutritious, and the bowels should be kept active. If, from advancing disease, mucus accumulates in the bronchial tubes, carbonate of ammonium will sometimes clear the chest in a remarkable manner, but may, of course, have only a temporary effect.

In the later stages, tonics—such as quinine, arsenic, iron, strychnine—should be given. If the limbs are flaccid, galvanism or faradism, massage and passive movements, may be of service; but they are of less value, or not advisable in cases of rigidity, well-nourished muscles, and increased reflexes, when their use may unduly excite the reflex action of the muscles.

CHRONIC MYELITIS

The occurrence of an inflammation of the spinal cord, chronic from the first, is believed to be very rare. Most cases so named have been, no doubt, instances of irrecoverable thrombosis and softening. And some other cases have proved to be disseminated or insular sclerosis. Whether the *scleroses* involving the different tracts of the spinal cord and constituting the lesions of locomotor ataxy, spastic paraplegia, and allied disorders should be regarded as chronic myelitis is questionable; they are almost certainly primary degenerations of the nerve-fibres.

Whether inflammatory or degenerative, these chronic lesions may be transverse or focal, disseminated or diffuse. Cases believed to be primarily chronic have appeared to arise sometimes from syphilis, repeated exposure to cold, alcohol, or chronic lead poisoning. A chronic myelitis may follow upon spinal meningitis, and then affects the surface of the cord adjacent to the membranes, forming a *peripheral* myelitis.

Anatomically the lesions present themselves in the form of reddish or gray patches, of varying consistence, but often hard, and sometimes slightly shrunken. Microscopically there is an interstitial inflammation, resulting in a fibrillated or amorphous tissue, with numerous nucleated cells, oval, fusiform, or stellate.

The nerve-fibres are generally destroyed, and in the gray matter the nerve-cells may also disappear, or be reduced to small angular bodies. The arteries are thickened, and the interstitial tissue is often especially abundant around them. In recent stages granule-corpuscles may be found.

When the lesion is situate in a conducting path, secondary degenerations take place upwards or downwards, according as a motor or sensory path is involved.

Symptoms.—These vary, in different cases, with the localisation of the lesions, and resemble those of the acute forms. A transverse lesion causes paralysis, with some anæsthesia, often very little, the whole developing in the course of months or years; ultimately excessive reflexes and spastic rigidity supervene. The bladder also is generally affected. If the two sides of the cord are affected unequally, one leg is more paralysed than the other, or even a unilateral lesion may exist with paralysis in one leg and anæsthesia in the other. If the cervical or lumbar region is affected, paralysis and anæsthesia may be accompanied with muscular atrophy, from implication of the nerve-centres of the brachial and lumbar plexuses respectively; and with this there will be some loss of electrical irritability, or even the degenerative reaction may occur.

Diagnosis.—Many cases are distinguished by the irregular way in which the symptoms are grouped. A localised transverse myelitis may be confounded with compression by tumour or caries, or with primary spastic paraplegia. In compression there is generally more evidence of irritation, and other symptoms due to tumour or spinal disease may be detected. Primary spastic paraplegia is distinguished by the absence of sensory symptoms, though the motor conditions may be closely similar in the two diseases. More diffuse forms may resemble pachymeningitis or progressive muscular atrophy: in the former there is more anæsthesia, and often more pain in the back; in the latter, sensory symptoms are absent.

Treatment.—The disease is little amenable to treatment, but is sometimes arrested. The most efficient means are rest, change of air, tonics, and the use of counter-irritation by mustard plaster, blisters, or even the actual cautery.

Brown-Séquard recommended a hot douche to the back at a temperature of 100° to 104° F. Gowers recommends, as drugs, arsenic, small doses of the red iodide of mercury ($\frac{1}{24}$ grain), and iodide of iron; and is of opinion that neither mercury in large doses nor potassium iodide does any good. The same complications will have to be treated as in the acuter forms or earlier stages.

SENILE PARAPLEGIA

In people of advanced age walking may become slow and difficult from weakness of the lower extremities. The gait is rather shuffling and the feet are dragged, difficulty is found in going upstairs, or getting into a carriage, and undue fatigue is experienced after any

exercise. The conditions may develop rapidly, and may go on to some stiffness and even contracture. It may be accompanied by slight sensory symptoms, pain or numbness, by impairment of the vesical sphincter, by senile trembling of the hands or head, by failing mental power, and by evidences of arterio-sclerosis. It is attributed to thrombosis and sclerosis of the vessels supplying the spinal cord in its lower part. Some improvement of the symptoms may be obtained from rest, massage, douches to the spine, and tonics.

LANDRY'S PARALYSIS

(*Acute Ascending Paralysis*)

In 1859 Landry described cases of paralysis commencing in the lower extremities, rapidly ascending, and soon fatal, for which no pathological cause could be found on examination. Improved histological methods and the science of bacteriology are throwing some light on these cases.

Ætiology.—The disease affects males more than females, and is most frequent between the ages of twenty and forty. It has occurred after exposure to cold, and in convalescence from acute diseases, in patients addicted to alcohol, and after syphilis, and a few cases have been recorded after cystitis or other forms of urinary sepsis.

Symptoms.—In some cases there are premonitory symptoms, such as malaise, pain in the head and back, and numbness and tingling, but usually the disease begins with weakness in the legs, often one before the other. This soon increases to marked paralysis, and invades successively, and within a few days, the thighs, trunk, abdomen, and arms ; and these, like the legs, are not always affected simultaneously. The diaphragm, and the muscles of the neck, of the palate, and those subserving articulation are subsequently paralysed. Very rarely other cranial nerves are affected : thus, diplopia, paralysis of accommodation, dilatation of one pupil, and facial paralysis have been noticed. The sensory functions are much less profoundly disturbed, but there may be, beside subjective sensations, slight anæsthesia, or hyperæsthesia, or tenderness of the muscles. In rapidly fatal cases the muscles have not wasted, and the electrical reactions have appeared to be normal ; but in some cases of longer duration both atrophy of muscles and modifications in electrical properties have been observed. The sphincters are generally active, there is no tendency to bed-sores ; the cerebral functions are perfect, and there is no fever, except in a few cases at the very onset. The knee-jerks are always absent, as are also the cutaneous reflexes in most cases.

The mortality is high—*e.g.*, 58 per cent. in cases collected by Ross. The duration of the disease is from two days to two or three weeks in fatal cases, and death occurs mostly from paralysis of the diaphragm and intercostal muscles. On the other hand, the symptoms persist

from two to six or seven months in cases which recover; but recovery is generally complete.

Pathology.—In a few, even recent, cases the spinal cord, nerves, and muscles have been found completely free from disease. In others there have been varying degrees of degenerative change (chromatolysis and displacement of the nucleus) in the cells of the spinal cord, especially those of the anterior cornua, with more or less vascular engorgement; and changes in the myelin of the anterior roots and of the white columns of the cord. In others inflammatory or degenerative changes in the peripheral nerves have been seen, and, in a few, changes have been found in the brain. Micro-organisms, but not always the same, have been found by different observers in the nerves, spinal cord, meninges, and blood. A tetracoccus has been isolated in two cases, in one after death, in the other during life from the blood, and from the fluid drawn by lumbar puncture. The spleen is enlarged in some cases.

All the more recent work points to the probability that the symptoms are due to toxins, of different degrees of virulence, operating upon the spinal cord and peripheral nerves, especially involving the lower motor neurons, sometimes producing such rapidly fatal effects as to leave but little trace, in other cases leading to degenerative or inflammatory tissue changes. Whether the micro-organism is one and the same in all cases it is at present impossible to say.

Treatment.—This may be the same as that of multiple neuritis or acute myelitis.

ACUTE ANTERIOR POLIOMYELITIS

(*Infantile Paralysis. Atrophic Spinal Paralysis*)

This is a special form of acute myelitis, in which the inflammatory change is almost entirely limited to the anterior cornua of gray matter, from which fact it derives its name (πολιός, gray).

Ætiology.—The most striking feature is the frequency with which it affects young children; it occurs six times more often in the first ten years than in the remainder of life. It has been attributed to cold and to falls, but there are many facts in favour of its being of an infective nature. It has occurred during convalescence from acute diseases, immediately after attacks believed to be influenza, and in women during the puerperal state. In a case under my care it began actually during confinement. Further, epidemics have been observed affecting a family or many persons in a community at the same time; and in the last few years outbreaks of considerable extent have occurred in Norway, Holland, Westphalia, New York, and Melbourne. In these last it has been thought that infection takes place by the naso-pharynx, and that the period of incubation is from one to two weeks. It must, however, be admitted that in these epidemics the symptoms have presented much more

diversity than is common in the familiar infantile paralysis, and their identity with it has perhaps yet to be established.

Symptoms.—The onset of the illness is often sudden, but the paralysis is not generally noticed for one, two, or three days. The early symptoms are different in different cases: there may be feverishness, or convulsions, or severe pains, either general or localised to the limb or limbs that are afterwards paralysed; or the child may go to bed well, and be found paralysed in the morning.

Both pains and feverishness are often present together, and the pain may persist some days. The way in which the paralysis itself begins is also variable: it may show itself in one limb, and within two or three days affects others; on the other hand, sometimes three or four limbs are paralysed at first, and recovery rapidly takes place in one or two, leaving the others permanently affected; finally, in other cases, certain limbs are affected from the first and remain so. A child, aged four and a half, was sick one day, feverish with headache the next two days, and became paralysed in all four limbs on the fourth. Three months later paralysis of both arms and weakness of the right leg remained. An adult had acute pains all over him, with general and severe prostration; after thirty-six hours he was paralysed in the lower extremities, the right being less affected than the left. A child, aged fifteen months, was noticed one day to have something wrong with the hip. She was feverish for three days; then the left leg was found to be painful and paralysed. The pain subsided in ten days, but the paralysis persisted.

I have hitherto spoken of limbs being affected, but it is characteristic of the disease that it need not affect the whole of a limb, but often only a part, or even one muscle: thus the upper arm, or the forearm, the anterior tibial muscles, the muscles of the thigh, or some muscles of one limb with some of another, may be paralysed; and even if paralysis affects the two legs, or the arm and leg on one side, it generally wants the uniform distribution of ordinary paraplegia or hemiplegia. On the other hand, the muscles of the trunk, abdomen, or neck are sometimes involved.

The affected muscles rapidly undergo atrophy, lose bulk, and become flaccid; when tested electrically they show the reaction of degeneration (*see* p. 210), or in severe cases do not respond at all to either current. The reflexes are quite lost, the knee-jerk disappearing when the lower extremities are involved. Sensation is never seriously affected, though there may be pain, tingling, or formication in the early stage. The bladder and rectum are always unaffected. After the first few days there is no further change for the worse in the extent or distribution of the paralysis. No fresh paralysis occurs, and any improvement of the affected muscles takes place with extreme slowness. According to the number of muscles atrophied will the use of the limbs be impaired; but after a time, in many cases, lost movements are restored by fresh combinations among the muscles that have been spared. Atrophy is, in almost all cases, a prominent feature, hollowing out the rounded part of

the forearm, or reducing the upper arm or the leg to a mere stick. Sometimes, however, the loss of muscle may be entirely concealed by the presence of fat; the flabby condition of the muscle even then can be generally recognised. Associated with the atrophic condition of the muscles is generally a change in the vascularity of the limb; it is cold, shrunken, and blue or livid from retarded circulation. The nutrition of the bones and other parts is also involved, so that a limb paralysed in infancy or early childhood does not grow with the same rapidity as its fellow, and may be shorter by half an inch, an inch, or more. Lastly, deformities occur besides those directly due to loss of muscular substance. Some are the simple result of failing muscular support; thus, from atrophy of the deltoid, the humerus falls from the glenoid cavity. Others consist of permanent changes in the position of the limbs, such as talipes equinus, which so often results from paralysis of the anterior tibial muscles. This was long attributed to the unresisted contraction of the calf muscles, but it has been shown by Volkmann that the chief element in its causation is the weight of the foot, which, whether the patient is upright or recumbent, falls into a position of extension unless supported by the anterior tibial muscles. The position thus constantly assumed becomes fixed by connective-tissue changes, both in the shortened calf muscles and about the ankle-joint.

In the epidemic variety, now often called *Heine-Médin's disease*, the lesions are not confined to the spinal anterior cornua, but may involve as well the medulla, pons, cerebrum or cerebellum; and the symptoms will correspond to the varying situations (*see* Myelitis, Bulbar Paralysis, Acute Encephalitis). The paralytic symptoms are also often accompanied by signs of irritation, such as pain, spasm, and Kernig's sign, suggesting extension to the meninges.

Prognosis.—In its sporadic form the disease is rarely fatal, as long as it is limited to the spinal cord: but its extension to the medulla oblongata adds a danger to life by causing an acute bulbar paralysis; and complete recovery is rare. Nearly always one or more of the affected muscles remains atrophied. Improvement in locomotion, or the use of the arms, may go on for several months, but even then it is obvious, from the outline of the limb, a limping gait, or a swinging foot, that a great deal remains uncured. The mortality in the epidemics has, however, been very high, reaching 15 or 20 per cent.; on the other hand, the proportion of cases of complete recovery is higher than in the sporadic form.

Morbid Anatomy.—Several cases have been examined after one or two years' duration, but quite early cases are less numerous. Drummond found in a case, presumably of this nature, which was fatal in a few hours, redness of the anterior gray cornua, arterioles and capillaries distended with blood, minute extravasations of blood, and swelling of the neuroglial elements and of the ganglion-cells. In cases of five or six weeks' duration, vascular distension, minute hæmorrhages, disappearance of the ganglion-cells, and degeneration of the anterior roots have been observed.

In later stages, the cord presents changes obvious to the naked eye: the motor nerve-roots, coming from the part presumably affected, are diminished in size and number. On a transverse section, the cord is smaller on the affected side, and the anterior cornu is shrunken. Under the microscope, there is an almost entire absence of motor nerve-cells and axis-cylinders; the few nerve-cells that remain are smaller than normal, shrunken, fusiform, and wanting in processes, and lie in a dense felt-like connective tissue. The motor nerve-roots, both in and beyond the cord, show the destruction of axis-cylinders, and are obviously degenerated.

Thus, the lesion appears to be mainly an acute inflammation of the anterior cornu, followed by sclerotic changes, involving the destruction of lower motor neurons with the necessary results in nerve-fibres and muscles. The last are pale-pink, watery in appearance, and present under the microscope the changes described as the result of lesions of motor nerves (*see* p. 214). But the view that it is necessarily an inflammatory lesion is not held by all. Gowers suggests that in some cases a hæmorrhage may be the primary cause, and others believe that it is frequently an acute softening due to thrombosis of vessels. Allen Starr would admit each of these conditions in different cases: and there is an obvious analogy with encephalitis. Several observers have isolated organisms (diplococci, tetracocci, meningococci, and staphylococci) from the cerebro-spinal fluid drawn off by lumbar puncture. W. Pasteur caused paralysis in rabbits by inoculating them with a patient's cerebro-spinal fluid which contained diplococci.

In Heine-Médin's disease emulsions of the affected nerve-tissues injected into monkeys have caused a corresponding disease; but no specific organism has been recognised.

Diagnosis.—In the early stages it is possible to mistake it for other acute affections of childhood, such as pneumonia, or meningitis. The pain that is sometimes present may suggest *rheumatism*, but it is situate rather in bone and muscle than in the joints. When the paralysis develops, within two or three days the diagnosis is generally clear, and it is confirmed by the rapid atrophy, the loss of reaction to the faradic current, and the changed reaction to galvanism. In cases of old standing, while the muscular atrophy, with retained sensation, indicates a lesion of the anterior cornu, the history of acute onset distinguishes the lesion from others less acute, such as *progressive muscular atrophy*. It is important not to be induced, by the term "infantile paralysis," to think that other forms of paralysis never occur at an early age. Paraplegia may result from spinal disease, or a tumour of the cord; and hemiplegia occurs from embolism, or congenital lesions of the brain. In these cases there is not the same rapid atrophy of muscles, and the electrical reactions are normal because in them the upper motor neuron is chiefly involved, and not the lower.

Treatment.—In the earliest stage, before the paralysis has developed, attempts may be made to reduce the fever, but the

treatment must be mainly expectant. When the paralysis has declared itself, the patient, already in bed, may be placed in the prone or lateral position, and ice-bags may be applied to the spine. Ergotin, in doses of $\frac{1}{6}$ to $\frac{1}{2}$ grain for children, and 1 or 2 grains for adults, twice daily, and belladonna, in doses of $\frac{1}{20}$ to $\frac{1}{6}$ grain of the extract, according to the age of the child, have been recommended. The patient will be generally benefited, after the first few weeks, by the use of tonics, such as iron iodide and phosphate, small doses of arsenic or strychnia, and cod-liver oil. At this time also the local treatment of the atrophied muscles becomes important. Electricity, in the form of faradism, to those muscles which are still susceptible to it, and galvanism to others, should be employed. Massage is also of value in promoting the circulation of the limb.

Deformities in the affected limbs must be prevented, as far as possible, by position and passive movements, and the latter should be employed early. Mechanical supports, and in some cases the division of tendons, may be necessary. A paralysed muscle which is unduly stretched by the weight of the limb, or by over-action of its antagonists, may be aided by fixing the limb for long periods of time so as to prevent this. Some assistance has also been gained by dividing the tendon of a paralysed muscle and grafting it on to the adjacent tendon of a healthy muscle.

CHRONIC ANTERIOR POLIOMYELITIS

Subacute and *chronic* forms of atrophic spinal paralysis have been described; but it seems that some of these must have been instances of peripheral neuritis. True subacute cornual myelitis occurs in adults more often than in children. The legs are first affected with paralysis, and afterwards atrophy; but instead of the symptoms reaching their maximum in a short time, the atrophy progressively increases for months or a year or two. It may then become stationary, and ultimately much improvement or recovery may follow; but in some cases death results from respiratory paralysis. In the altered electrical reactions, on the one hand, and in the normal condition of the sensory functions, and of the bladder and of the rectum, on the other, it resembles the acute form of the disease. It commonly affects the lower extremities first, and only after a considerable period attacks the arms.

HÆMORRHAGE INTO THE SPINAL CORD

This is a very rare occurrence, and contrasts remarkably with hæmorrhage into the brain, which is one of the most common causes of cerebral paralysis.

Ætiology.—It occurs in younger persons than does cerebral hæmorrhage, and in males more often than in females. The chief causes are: (1) Injuries, falls upon the feet, strains, &c.; these

form nearly 90 per cent. of all cases of hæmorrhage, and the cervical region of the cord is the part most commonly affected; (2) alterations in the vascularity of the cord, or structural changes in the walls of its blood-vessels; (3) a preceding lesion of the cord, such as a soft gliomatous growth, the vessels of which may rupture, or, perhaps more often, an acute myelitis in its early stage. A primary hæmorrhage is generally confined to the gray matter, and is of small extent, rarely exceeding the size of an almond; but in cases of congestion it is punctiform, and may occupy both white and gray matter. It is also more diffused when secondary to myelitis. Gowers records cases of hæmorrhage into the cavity of congenital syringomyelia: if this is abundant it will compress or tear up the tissues of the cord.

Symptoms.—The onset is often quite sudden: the patient may be seized with acute pain in the back, and then fall, with complete paralysis of motion and sensation below the seat of the lesion. In other cases the symptoms may be more gradually developed in the course of a few hours. The limbs are mostly relaxed, and there may be clonic contraction in the muscles, either immediately or in a few days. The symptoms are subsequently those of an acute local myelitis—paralysis, loss of sensation, the reflexes increased after a short period, during which they are diminished, and the bladder affected. With a central hæmorrhage there may be dissociation of sensations (*see* Syringomyelia); and with hæmorrhage into a syringomyelic cavity sensory effects may be more marked than motor, because the cavity so often occupies the posterior part of the cord. There may be elevation of temperature after a few days from secondary inflammation, and this myelitis may spread upwards and downwards. Secondary degeneration of the lateral and posterior columns frequently follows, and accompanying this is spastic rigidity of the limbs, with increased knee-jerk and ankle clonus; but if the gray matter is much destroyed in the cervical or lumbar regions, wasting of the corresponding muscles may supervene. Trophic changes, cystitis, and bed-sores are also not infrequent.

Diagnosis.—This depends on the sudden onset of the symptoms, but the disease may be confounded with a *hæmorrhagic myelitis*, and with *meningeal hæmorrhage*. Prodromal symptoms of even very short duration, and fever, make myelitis or softening probable. *Meningeal* or *extra-medullary hæmorrhage* is distinguished by the signs of nerve-irritation rather than nerve-impairment, such as severe pains in the distribution of certain nerves, and muscular cramps, as compared with anæsthesia and paralysis in spinal hæmorrhage. The bladder is less likely to be affected, bed-sores do not occur, and the disease is less fatal. *Acute anterior poliomyelitis* begins as an acute myelitis, and it is even possible that there is in some cases hæmorrhage, but the symptoms are less sudden. The absence of back-pain, the initial fever, or convulsion, the freedom from vesical, rectal, and sensory symptoms, and the rapid localised atrophy, readily distinguish this disorder from spinal hæmorrhage.

The **Prognosis** is unfavourable; many cases are fatal; and others develop into conditions of permanent chronic paralysis, like cases of myelitis. In some instances there is rapid recovery up to a certain point, with no further improvement.

Treatment.—The patient should be placed in the prone position, if possible, or on the side, to prevent the spinal cord being in the lowest part of the body. Ice-bags should be applied to the spine; and if symptoms are progressing, blood may be withdrawn by leeches or cupping to the back, or leeches to the anus. Ergotin should be injected in doses of three grains, every two or three hours, up to three or four doses, or the liquid extract of ergot may be given by the mouth. Pain may require to be relieved by sedatives. Later on the treatment is similar to that of myelitis.

CAISSON DISEASE

(*Divers' Paralysis*)

Divers and those who work in caissons at great depths below the surface of the earth, such as are equivalent to a pressure of three or four atmospheres, are liable to a form of paraplegia, which supervenes on their return to the surface; and this is more likely to occur if they are brought up quickly than otherwise. The symptoms may be mild or severe, and come on within an hour of the changed conditions. In mild cases there is only a little weakness with numbness of the lower extremities, which passes off in a few hours or days; in other cases the arms are also affected, or the symptoms are more severe and last several weeks; in others again there is coma, and the patient may die in it. Some persons suffer more often from less serious symptoms, such as pains in the ears, deafness, giddiness, severe pains in the legs, arms, and shoulders (called by them the *bends*), nervousness or intense excitement, and bleeding from the nose, mouth or lungs. These, with the paralysis, constitute *caisson disease* or *compressed air illness;* and they are all due to a too rapid *decompression* or return to normal pressure.

The immediate cause of the paralysis appears to be that in consequence of the pressure an excessive quantity of gas is forced into the blood and tissues, and that on return to the upper air, the gas escapes more rapidly from the tissue-cells than the blood can carry it away: and thus bubbles are formed which press upon the tissues. In the spinal cord this affects its functions seriously. Fissures have been found in the cord, filled with leucocytes, and surrounded by small areas of inflammation; hæmorrhages are sometimes present.

For the prevention of the symptoms it appears to be necessary that the pressure under which the men have worked shall be gradually reduced to the normal (by suitable apparatus in an intermediate chamber or *lock*) at a rate not exceeding one pound in three minutes; and the symptoms may be removed by the patient

immediately returning to the lock, undergoing *recompression*, and only coming out again after *decompression* at the standard rate. In most cases, however, the paralysis recovers in time, and should be treated on the same lines as cases of myelitis. For severe pains injections of morphia may be given; and in some instances the catheter will be needed to empty the bladder.

TABES DORSALIS

(*Locomotor Ataxy*)

Tabes dorsalis is essentially a chronic degeneration, with sclerosis, of the posterior columns of the spinal cord (posterior sclerosis); with similar changes in the posterior roots, in the peripheral nerves, and sometimes in the optic nerves and other structures. The chief clinical feature is locomotor ataxy, or inco-ordination of movements in walking, while the muscles retain full power of contraction; but this is both preceded and accompanied by various disturbances of the nerves of sense, sensation, and nutrition.

Ætiology.—Syphilis is undoubtedly the chief factor in the occurrence of this disease, which is included in the group of para-syphilitic disorders. A history of syphilis is present in from 70 to 80 per cent. of adult cases of tabes, and nearly all juvenile cases are children of syphilitic parents: moreover, the Wassermann reaction (*see* p. 143) has been obtained in numerous instances. Cold and wet, injuries to the spine and sexual excesses may act by depressing the general vitality.

The disease arises mostly in the middle period of life, between twenty and fifty; but many cases in younger individuals have been recorded. In adults it is much more frequent in men than women, in the proportion of ten to one; but in juvenile cases females have been more numerous, in the proportion of three to two.

Symptoms.—In spite of the limitation implied in the clinical name, locomotor ataxy, there is scarcely a disease in which the functional nervous disorders are so numerous and widespread; with the result that in different cases different symptoms may be more prominent, and lead to errors in diagnosis unless their connection with the spinal lesion is familiarly known.

In the early (pre-ataxic) stage the characteristic symptoms are pains in the limbs, loss of knee-jerk, and loss of pupil light reflex. This stage may last for months or years.

The pains known as *lightning pains* occur in 95 per cent. of the cases; they are severe shooting, stabbing, or darting pains in the lower extremities, sometimes resembling electric shocks. They are often looked upon as rheumatic, but they are seated in the muscles and bones, and not in the joints. They come on suddenly, and it may be with such severity as to make the patient start up in bed, or cry out. They may subside in a few minutes, but generally soon recur, and continue thus, coming and going, for several hours.

They may then disappear, and not return till the next day, or after an interval of days or weeks. They thus present the greatest irregularity both as to recurrence and duration.

The *knee-jerk is abolished* quite early, and this is one of the earliest and most common symptoms of tabes, occurring in more than 80 per cent. of the cases. The *Achilles jerk* is also very frequently absent. The plantar reflex is sometimes absent, at others present or even exaggerated.

In more than four-fifths of the cases the pupil fails to contract to the stimulus of light, though it continues to contract during accommodation for near vision (*Argyll-Robertson pupil*). In addition to this, there is often, quite early, slight *anæsthesia* of the feet and lower part of the legs, and occasionally temporary paralysis of one or more of the ocular muscles, leading to diplopia, or squinting, or ptosis, according to the muscle involved.

In the second stage, that of the developed disease, or actual locomotor ataxy, the prominent feature is the *muscular inco-ordination* of the lower extremities, and this is associated with increased anæsthesia and other sensory disorders, and impairment of the functions of the bladder. Other rarer conditions, which are generally first observed in the early stage, are the so-called gastric and other *crises;* certain trophic disturbances; optic-nerve atrophy, myosis, and other ocular conditions.

The *ataxy,* as indicated by the epithet "locomotor," is chiefly and first noticed in the lower extremities, and is confined to them in a great number of cases. At first the patient is only slightly unsteady in his gait, finds a difficulty in walking quite straight, separates the legs a little to meet this difficulty, keeps his eyes carefully fixed on the path he is walking, and readily loses his balance when trying to turn. In the dark, when the guiding sensations of sight are removed, he is still more unsteady. If Romberg's test be employed, the patient staggers or falls (*see* p. 201). In a later stage, walking can still be accomplished, but the legs are drawn up or jerked up in a disorderly way; they are often thrown violently forwards, and the heels are brought down with force upon the ground. Turning is still more difficult than before, and has to be effected with great care and the assistance of a stick, a wall, or a friend. Movements may also take place when the patient is quiet (*static ataxy*). Nevertheless the muscular power remains good. The patient can bear another man on his back, and if he sits in a chair he can keep his leg extended in front of him against any ordinary attempts of the medical man to flex it. Moreover, the muscles are of normal bulk, and give the normal electrical reactions. The distance which the patient can walk is lessened to a mile or two, on account of the great waste of strength involved in these disorderly and, therefore, ineffective movements. In later stages the ataxy may be such that he cannot walk at all without assistance from sticks, a chair, or a friend on either side; and, finally, he may have to take to his bed.

The arms are sometimes affected late in the disease, but the ataxy is generally less extensive than that observed in the legs.

Hypotonia is another result of the loss of reflex muscular tone, already shown in the absence of tendon jerks. There is in this condition a remarkable mobility of the limbs, so that joints can be passively flexed or extended to quite abnormal degrees without the resistance and pain which are caused in healthy individuals. Frenkel states that hypotonia is a constant symptom even in the earliest stages.

Anæsthesia is variable. It affects the feet and legs, spreading to the knees, or even to the thighs and buttocks, and sometimes to the trunk. If the upper extremities are affected, it begins in the fingers and hands, creeping gradually up the forearms. The loss of sensation gives rise, when the patient is standing or sitting, to a peculiar feeling of being on some soft substance, which patients describe as being like water, wool, sponge, or india-rubber. Numerous other modifications of sensation are observed in different cases—burning or gnawing pains in the extremities, more continuous than the lightning pains; a sense of constriction in the legs, groins, genitals, or trunk, the latter often described as "girdle-pain"; tingling, pins and needles, sensations of cold or heat, and increased sensitiveness to alterations of temperature.

The anæsthesia may take the form of diminished sensibility to pain alone, or to touch alone, or differences of temperature may be less readily perceived. In some cases the perception of pain or of heat and cold is delayed, or the pain recurs after the source of it has been removed, or the allochiria or polyæsthesia above described (*see* p. 202) is present. Absence of pain on pinching the ulnar nerve at the elbow has been called *Biernacki's sign.* The deeper tissues are also anæsthetic, certainly the muscles, and probably the joints, fibrous tissues, and tendons. The *loss of the muscular sense* is an important part of locomotor ataxy, and may be shown by the patient's incoordinate movement, and ignorance of the position of his limbs when his eyes are shut. The sense of muscular contraction is defective; the muscles bear pressure without discomfort, and are less sensible than normal to the faradic current. In many cases there is insensibility to passive movements of the joints. Another condition, often present and probably allied to the above, is defective sensibility to the vibration of a tuning-fork (*pallæsthesia*).

The *bladder* is often affected as follows: In early stages, there is irritation, with frequent micturition, and the necessity of passing urine directly the desire is perceived. Later on, the detrusor is weakened, and the urine comes in a sluggish stream, or merely dribbles away. Sometimes there is retention with incontinence from overflow. The *sphincter ani* is generally weakened, or fæces are passed unconsciously from insensibility of the anus. Sexual power is commonly lost.

The curious functional disturbances of the viscera, called *crises*, occur in about one-fifth of the cases. The *gastric crisis* (*crise*

gastrique) is the most frequent; in each attack there is severe pain in the epigastrium, passing through to the back, or extending from the groins up to the shoulders, accompanied by vomiting, at first of clear liquid, later of bile, and even of blood. Pain may occur without vomiting, or vomiting without pain. Often, also, there is palpitation or irregularity of the heart. These symptoms last for two or three days, and then subside, leaving the functions of the stomach quite normal. The other *crises* that have been described are *rectal crisis*, consisting of paroxysmal pain in the rectum with severe tenesmus; sensations referred to the genital organs—*sexual crisis;* paroxysmal diarrhœa, or *intestinal crisis;* paroxysms of renal pain, or *renal crisis;* pain in the bladder or urethra—*vesical* or *urethral crisis; laryngeal crisis*, consisting of laryngeal spasm, with inspiratory and expiratory stridor, cough, and dyspnœa; and *nasal* or *bronchial crisis*, when there are paroxysms of sneezing or coughing.

The *trophic* disturbances which occasionally occur in tabes dorsalis are œdema of the feet, local sweating, ecchymoses under the skin, brittleness of the hair, and herpes, the last three in connection with severe pains. The skin of the sole of the foot becomes thickened, or blisters, or may present a deep circular and conical depression, the *perforating ulcer*. The nails become thickened and furrowed, or fall off, and are slowly renewed. Teeth decay, or may fall out within a short time.

In occasional cases, about 6 per cent., important changes take place in the *bones* and *joints*. The bones become brittle; the compact tissue has been found thinner and more porous; fractures occur spontaneously, or with the slightest amount of force; and a great deal of callus forms in the process of union. The lesions in the joints are known by the name of *Charcot's disease*. The changes are almost identical with those which occur in osteo-arthritis, or rheumatoid arthritis, namely, erosion of cartilage, wasting of the head of the bones, ossification of the ligaments, and new bony outgrowths. Clinically, they are characterised by rapid painless swelling from effusion into the joint, and subsequently extreme mobility and grating. While some hold that these changes are the direct result of the withdrawal of trophic influence from the part, others consider them to be due to the external injuries, strains, &c., which ataxic limbs are so much more likely to suffer than healthy ones.

In addition to the loss of light-reflex the pupils may show other disturbances, such as inequality, extreme contraction, irregularity of outline, eccentricity of position, failure to dilate on pinching of the skin, or failure to contract with accommodation. Primary (gray) atrophy of the optic nerve occurs in about one-fifth of the cases, starting on the temporal side; this results in contraction of the visual field from the periphery inwards, and loss of vision for colours (*dyschromatopia*) in the following order: green, red, yellow, blue, and violet.

Deafness, paralysis of the abductors of the vocal cords, severe

headache, glycosuria, and apoplectiform or epileptiform attacks occasionally occur.

Course of the Disease.—The symptoms are often stationary for very long periods, and the disease may last twenty years or more. Even the pre-ataxic stage may persist for several years;

FIG. 28

Illustrates arthropathy (Charcot's joints) of the right knee and ankle in a case of tabes dorsalis. (*After Turner and Stewart.*)

and, on the other hand, sufferers who are unable to leave their beds may live to old age. Optic atrophy may be pre-ataxic, accompanied only by Argyll-Robertson pupil, absence of knee-jerks, and some pains. Such cases often progress slowly, or even improve for a time. Similarly, gastric crises may occur long before any ataxia is present, but with the same associates as above. The majority of patients die, not from the disease itself, but from intercurrent affections, such as pneumonia, phthisis, bronchitis, apoplexy, or other independent ailment. Cystitis and renal complications, bed-sores and pyæmia, and rarely laryngeal spasm, are direct consequences of the disease, and may be fatal. Some cases

terminate in general paralysis of the insane, a cerebro-spinal disorder resulting from syphilis, in exactly the same way as tabes dorsalis.

Morbid Anatomy.—The change constantly found in the spinal cord is a degeneration of nerve-fibres and sclerosis of neuroglia in the posterior columns. It is seen as a gray discoloration of the white matter, and is brought out more readily by the hardening and staining processes. In ordinary cases it occupies in the lumbar region the whole of the posterior columns, but above the lumbar region the sclerosis of the postero-external columns ceases, whereas the sclerosis of the posterior median columns continues up into the cervical region. The lesion of the postero-external column is most intense in its posterior portion, the anterior portion near the commissure being sometimes free. In very severe cases involving the arms, the postero-external columns may be sclerosed in the higher parts of the spinal cord as well as in the lumbar region. Lissauer's tracts are said to be early affected; and the antero-lateral ascending tract and the cerebellar tract are occasionally degenerated. Under the microscope, degeneration and disappearance of the nerve-fibres, increase of the connective tissue, which is fibrillated, and thickening of the walls of the arteries, are observed. In advanced cases changes may also be detected in the gray matter, such as atrophy and degeneration of nerve-cells or fibres in the posterior horns, in Clarke's columns, in the tractus intermedio-lateralis, and even in the anterior cornua. The pia mater is often thickened over the posterior columns, or even over the lateral columns as well, or completely round the cord. The cerebro-spinal fluid, as may be shown by lumbar puncture during life, contains an excess of lymphocytes.

The posterior nerve-roots are generally atrophied down to the spinal ganglia, which are mostly healthy, as well as the mixed nerves beyond them; but atrophy of the peripheral nerves has been also found, mostly of those supplying the skin and the joints, and in the legs more commonly than in the arms. In the optic nerves, in cases alluded to, are found atrophied nerve-fibres and increased connective tissue. Atrophy or degeneration of the nuclei of the third, fourth, fifth, sixth, eighth, and twelfth cranial nerves has in different cases been seen; as well as of the Gasserian ganglion; but the lesion of reflex iridoplegia in tabes is not certainly known, though Marina has found degenerative changes in the ciliary ganglia and short ciliary nerves.

Pathology.—As there is no gummatous change present in tabes dorsalis, the lesions can only be ascribed to a toxin which must cause primary degeneration of the nerve-fibres. This is followed by a corresponding and proportionate sclerosis. The method of cyto-diagnosis (*see* p. 213) shows an excess of lymphocytes in the cerebro-spinal fluid, and this suggests persistent irritation of the surface of the cord. It is clear that the lesion begins below and ascends the cord; and that the sclerosis of the posterior median columns is secondary or consecutive to that of the postero-external columns

below. There is therefore a primary lesion in the *lower sensory neurons* (protoneurons) within the spinal cord, probably first of all in the terminal fibres which arboresce round the cells of Clarke's columns, and round the cells of the posterior column nuclei (Spielmeyer), but also in the reflex collaterals to the anterior cornua.

FIG. 29

Cervical. Dorsal. Lumbar.

Position of Lesions of Locomotor Ataxy in different parts of the Spinal Cord.

Other sensory neurons may be also affected, such as those of the cerebellar tracts, and the optic nerve. Thus are explained some early features of tabes dorsalis, *e.g.*, the pains, anæsthesia, the lost reflexes, and the hypotonia. Inco-ordination of muscular movement, though at first sight a motor derangement, may clearly be brought out by interference with the paths by which sensory impressions are conveyed to the centre. But ataxy does not depend necessarily on cutaneous anæsthesia, since either may exist without the other; and it probably depends upon loss or impairment of afferent impressions of all kinds, conscious as well as unconscious, and especially of those from the deeper structures, muscles, tendons, and joints, to the spinal, subcortical, and cerebellar centres (Ferrier). When a muscular effort is made, the defective conduction in these afferent paths leads to an absence of the reflex resistance of the analogous muscles and of the proper muscular adjustments innervated by the cerebellum; and these are only imperfectly compensated by the visual sense. The opinion that the actual commencement is in the synapses between the lower and the middle sensory neurons is supported by the fact, that although the primary neurons are alone diseased, the sensory disturbances of tabes are variable and irregular, and do not correspond to the definite lesions of epicritic, protopathic, and deep sensibilities which would be manifest in disease either of the posterior roots or of the peripheral nerves (H. T. Thompson).

Since the muscle-sensory nerves, equally with the cutaneous nerves, are contained in peripheral nerve-trunks, and the posterior roots, it might be expected that ataxy would sometimes result from lesion of these parts; and, as a fact, peripheral neuritis from alcoholism may cause ataxy; and a close resemblance to tabes dorsalis was seen in a case of multiple tumours of the posterior roots recorded by Hughes-Bennett.

Diagnosis.—Tabes dorsalis has to be recognised in its early

stages before inco-ordination is pronounced; and it has to be distinguished in the stage of ataxy from other disorders affecting the power of the lower extremities. The lightning pains are generally very characteristic, but the absence of knee-jerk and the loss of light-reflex of the pupil are the distinctive features. In a more advanced case the same two signs are of service, and in addition, the inability to stand with the eyes shut, or to turn with steadiness. Where locomotion is much interfered with, the case contrasts with *paraplegia* from myelitis, by the retention of absolute muscular power, and by the normal bulk of the muscles, with absence of rigidity. Where muscular wasting and weakness supervene there may be more difficulty in diagnosis, but the long history and the course of the symptoms will assist. *Cerebellar* disease also causes ataxia, but it is generally of a reeling, staggering kind, the patient swaying from side to side, falling over, crossing the legs to recover balance, and presenting a close resemblance to a drunken man; whereas, in the locomotor ataxy of tabes, for a time the gait may be steady in direction, but the feet are jerked forward, and the heel or flat of the foot is brought down sharply on the ground. The two diseases have also their special accompanying symptoms.

It is important to remember that a patient with gastric crises, a perforating ulcer of the foot, or "Charcot's joint," may be entirely unaware of any locomotor symptoms; and in such cases the knee-jerk and the pupils should be at once tested. Otherwise, a diagnosis might be formed, which would lead quite unnecessarily to a capital operation.

Peripheral neuritis in its ataxic form may be generally recognised by the atrophy and tenderness of the muscles, the "dropped foot," the high-stepping gait, the altered electrical reactions, the normal pupils, and possibly by an alcoholic history.

The cases of *combined sclerosis*, to be described shortly, in which the lateral columns are degenerated as well as the posterior, present spastic phenomena and muscular weakness, as well as ataxia of locomotion. The allied parasyphilitic disorder, general paralysis of the insane, occasionally commences with symptoms like those of tabes dorsalis.

Treatment.—This is by no means satisfactory; yet it is not uncommon for patients to be relieved of certain symptoms after a few months. Thus, pains disappear, anæsthesia diminishes, and incontinence of urine becomes less under treatment, although other features of the illness remain in full force.

The certainty that tabes dorsalis is essentially a syphilitic affection has led to a renewal of the specific treatment, which has until recently been regarded as quite useless. It now appears to be advisable to try a continuous course of mercurial inunction or potassium iodide in cases in which the symptoms are very recent—within four or five years—or in which the treatment of the original infection was admittedly inadequate.

The following drugs have also been employed at different times, but with little success : arsenic, strychnia, iron, quinine, belladonna, ergot, phosphorus, silver nitrate, and Calabar bean. The continuous current applied to the spine and legs has often seemed to me to do good by relieving some of the symptoms, if not improving the gait. For the pains antipyrin should be given in doses of 10 to 15 grains every three, four, or six hours, according to their severity. Aluminium chloride, 5 to 10 grains in water three times daily, is also of value ; and other remedies used are acetanilide, phenacetin, the salicylates, aspirin, and cannabis indica. In very severe cases morphia may require to be given, but should be avoided as long as possible. Attention to the bladder is very necessary, and the catheter should be used if any urine is retained. The expulsive power may be increased by strychnia, or incontinence may be lessened by belladonna. Gastric attacks may require morphia, or may be checked by blisters to the epigastrium. For the laryngeal spasm, Gowers recommends amyl nitrite and the local application of cocaine. In recent years it has been shown that, apart from the pathological condition underlying it, the ataxia itself can be very greatly diminished, or even temporarily cured, by graduated exercises (Goldscheider, Frenkel). The patient is conducted from simple and rhythmical to more complicated movements, and is made to pay such attention to them as to ensure a thorough re-education. The movements are active, and consist in flexion and extension of the legs to minimum, maximum, and intermediate extents while recumbent, sitting, or standing; in moving the foot from point to point in a regular, methodical manner; in walking slowly on marked lines, straight and zigzag, or in marked footsteps, forwards, sideways, and backwards, &c. They should be carried out three times daily, at first for ten or fifteen minutes, and later for half an hour to an hour. Massage may be usefully employed at the same time.

PRIMARY LATERAL SCLEROSIS

(Primary Spastic Paraplegia)

The condition in which weak or paretic limbs are rigid from spastic contraction of their muscles is known as *spastic paraplegia*, and is qualified as primary if it occurs spontaneously and independent of any such local disease of the cord as transverse myelitis, disseminated sclerosis, or pressure from tumour, aneurysm, or tubercular abscess. It is essentially a degenerative change in the pyramidal tract—that is, in the upper motor neurons situate in the spinal cord.

Ætiology.—The disease is not very common : it is more frequent in males than in females, and occurs mostly between the ages of twenty and forty. It has appeared in two or three members of

the same family. No adequate cause has hitherto been found; but it is certainly not caused by syphilis.

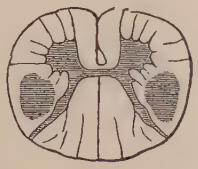
Symptoms.—The disease develops very slowly and insidiously. It begins with weakness and stiffness of the lower extremities; the legs feel heavy, the patient soon gets tired in walking, and, as time goes on, the distance he can walk without fatigue gets less and less. The knee-jerk is excessive; ankle-clonus and Babinski's sign are present; and the cutaneous reflexes are usually increased. After a time he has to help himself with sticks; the legs are rigidly extended and firmly abducted, and it is almost impossible to get one in front of the other. The rigidity is nearly constant, or, if the muscles relax slightly, they at once contract on a touch, or the stimulus of attempted movement. The arms are generally unaffected, but if they are involved they take up a position of rigid flexion. In some cases the muscles of the trunk are involved. The muscles mostly remain in good condition, and the electrical reactions are normal; sometimes, however, the excitability of the muscles is slightly increased, at others diminished, to both currents. Sensation is unaffected beyond the occurrence of rheumatoid pains and some tingling or numbness; and the bladder and rectum are in most cases free from the functional disturbances common in paraplegia and locomotor ataxy. The course of the disease is very chronic, and it may last twenty or thirty years; indeed, it is less threatening to life than are other forms of chronic spinal disease. It may be complicated in its late stages by extension to the anterior cornua, causing wasting of the muscles with loss of reflexes; or to the postero-external column, producing lightning pains and ataxy.

Pathology.—The pathological condition underlying the symptoms is a *sclerosis* of the spinal cord occupying the whole length of the pyramidal tract, or the posterior part of the lateral tract—hence a *lateral sclerosis*. The histological change is identical with that seen in the posterior sclerosis of tabes dorsalis, that is, degeneration and disappearance of nerve-fibres with increase of neuroglial tissue. Whether this is due to a congenital inability to survive, or to toxins, however produced, has yet to be known. Slight lesions of the cerebellar tract and of the columns of Goll may also be present, but do not appear to contribute to the symptoms.

Diagnosis.—The recognition of a spastic paraplegia is not difficult; weakness of the legs, spastic rigidity, increased reflexes and Babinski's sign are its characteristics; while sensation, the bladder and rectum, and cerebral and ocular functions are normal. The important point to be decided is whether the condition is *primary* or *secondary*, and very careful inquiries and examination should be made before concluding that a case of rigidity is not secondary to local disease of the spinal cord, such as transverse myelitis or softening, or compression. In primary disease of the lateral tracts, weakness and stiffness come on gradually and simultaneously;

whereas in secondary cases there is at first decided paralysis, rigidity only supervenes later, and sensory symptoms and girdle pain are often present. The same care will enable one to recognise the cases in which lateral sclerosis is combined with disease of other parts of the cord, as in amyotrophic lateral sclerosis or ataxic para-

Fig. 30



Position of Lesion in Primary Spastic Paraplegia.

plegia. Lateral sclerosis may also form a part of general paralysis of the insane.

A spastic paraplegia occurring in young children or infants is generally of cerebral origin (*see* Infantile Cerebral Diplegia).

Treatment by drugs is unsatisfactory. Arsenic, nitrate of silver, ergot, hydrobromic acid, potassium bromide, and potassium iodide have been given. Rest is very desirable. Friction of the muscles, combined with the hot bath or Turkish bath, or the application of hot bags to the spine, sometimes relieve the spasm. Electricity is of doubtful value; and, in general, it is desirable to avoid undue stimulation of the muscles in any way.

COMBINED POSTERIOR AND LATERAL SCLEROSIS

Under this heading are placed a number of cases, in which there are ataxic and sensory symptoms attributable to degeneration of the posterior columns, and spastic and paretic symptoms caused by degeneration of the pyramidal tracts. It includes the *ataxic paraplegia* of Gowers, and other cases called *spastic ataxia*.

Ætiology.—The variety in causation allows a division into several groups. There are those of which the cause is obscure, or entirely unknown; mostly there is no history of syphilis; a few have been referred to concussion of the spine. They are more common in men than in women, and the symptoms begin first in middle life. Secondly, these conditions, clinical and pathological, have been observed in association with pernicious anæmia and other profound anæmias and cachectic disorders. They are attributed to the action of toxins. Thirdly, spinal symptoms, which have in some instances been shown to be due to spinal sclerosis, occur in the disorders which arise from eating diseased grain, known as *ergotism* and *lathyrism*, as well as in *pellagra*, though in this instance the connection with diseased grain

is open to doubt. Fourthly, Erb has long described from the clinical point of view certain cases of spastic paralysis as a result of syphilis; and some of these have been shown *post-mortem* to present a combined sclerosis. Fifthly, the disease known as Friedreich's ataxy, or hereditary ataxy, is due to a combined sclerosis, probably the result of a congenital defect in the nutrition of the neurons concerned (*see* p. 291).

Symptoms.—Ataxic paraplegia, as described by Gowers, is slow in its onset. The symptoms begin in the lower extremities, which become weak, and show defective co-ordination in the unsteady reeling gait, and in the presence of Romberg's sign. The knee-jerk is increased and ankle-clonus is present; the plantar reflex is more often either normal or increased. Sexual power is often lost, and the sphincters may be slightly affected. But sensation is mostly unimpaired; the pupil and optic disc remain normal; and there are no lightning pains. The arms are more often free, but are sometimes involved in the same way as the legs. The disease is chronic in its course, with no very fatal tendency. As it progresses, the inco-ordination does not increase, but the weakness becomes more marked, rigidity occurs, and the resemblance to spastic paraplegia becomes close. Death results from intercurrent diseases, or occasionally from bladder complication or bed-sores.

But there is very considerable variety in the symptoms in the different cases, dependent upon the relative extent to which the posterior and the lateral columns are involved; and even in different stages, as the complete implication of the posterior columns abolishes the reflex phenomena associated with lateral sclerosis. If the lateral sclerosis is predominant, spastic paraplegia is well marked, accompanied it may be with ataxia, shooting pains, paresis of the bladder, loss of pupil light-reflex, and other tabetic symptoms: if the posterior sclerosis is in excess, there are the typical symptoms of tabes with muscular weakness. The implication of the pyramidal tracts may be here indicated by the occurrence of Babinski's sign.

In the cases associated with anæmia, the first symptom is often some paræsthesia of the feet: then follow weakness and ataxia, increased knee-jerks, ankle-clonus, and rigidity: and later the different forms of sensation are diminished. The duration of these cases is often less than three years, and is no doubt largely determined by the condition of the blood. In the more prolonged cases the arms may be affected, and paræsthesia, weakness, and ataxy are the first symptoms, as in the lower extremities. Paralysis of the diaphragm is an occasional cause of death.

The symptoms which Erb has associated with a syphilitic origin are spastic gait, motor weakness, rigidity of the legs, which is slight as compared with the spasticity of the gait, increased tendon reflexes, bladder troubles, and slight sensory disturbances.

The **Anatomical change** in this disorder is simultaneous sclerosis of the lateral columns and of the posterior columns, of varying extent and distribution, and differing slightly from what is common in tabes dorsalis and spastic paraplegia respectively. Thus, the

posterior sclerosis affects the dorsal more than the lumbar region, and leaves the postero-external columns intact, or does not reach to the surface of the cord. In the lateral columns the sclerosis tends to advance to the anterior half, instead of remaining limited strictly to the area of the pyramidal tract. The cerebellar tract is also

FIG. 31

Cervical. Dorsal. Lumbar.

Position of Lesion of Ataxic Paraplegia in different parts of the Spinal Cord.

involved, and occasionally Burdach's or Türck's column, or the antero-lateral ascending tract. Marie attributes the change to endarteritis of the spinal vessels entering the surface of the cord.

Diagnosis.—This has to be made in the early stages from *tabes dorsalis*, especially by the excessive knee-jerk, the normal pupil, and the absence of lightning pains, and in the later stages from *lateral sclerosis*, by the presence or history of inco-ordination. Later still, increasing paraplegia, with anæsthesia, and bladder symptoms, and diminution of the ataxy and rigidity, and loss of knee-jerk, would confirm the diagnosis. It may also be confounded with *hereditary ataxy*, and with disease (*e.g.*, tumour) of the *cerebellum*. In this last the muscular weakness is seldom so marked, and the local symptoms—headache and optic neuritis—should be sufficiently obvious. Some cases of ataxic paraplegia have proved subsequently to be due to *disseminated sclerosis*.

Prognosis.—This is very unfavourable; but in one case at least temporary improvement of the spinal symptoms followed treatment of the accompanying anæmia.

Treatment apart from this must be that of locomotor ataxia or primary spastic paraplegia.

HEREDITARY ATAXY

(*Friedreich's Ataxia*)

The characteristic feature of this form of combined sclerosis is its congenital tendency and its appearance in several members of the same family. Thus, a man may transmit it to his children, or it may appear in two or more brothers and sisters without the parents being affected. It affects males only a little more often than females, though males may be especially affected in one family, females in another; and it is generally first noticed at an early age, either about the seventh or eighth year, or at puberty.

Symptoms.—The disease begins with inco-ordination in the lower extremities, which soon involves the trunk; and the inco-ordination is generally more jerky than in other forms, and presents a closer resemblance to the ataxy of cerebellar disease. The arms are affected later with a similar jerky irregularity, and the same extends to the muscles of the head and neck, so that there is some resemblance to chorea. When the child is sitting still there is some oscillation of the head and body. Muscular power is at first unaffected; the knee-jerk is usually lost quite early, but Babinski's sign is often present. Sensation is often unaffected; sometimes there is slight anæsthesia. As a rule, lightning pains are not present. Speech is frequently impaired, as shown by hesitation; and syllables are dropped, and the movements of the tongue may be jerky. Nystagmus, or oscillation of the eyeballs, is often present; it is rather slow, lateral in direction, brought on by lateral movements, and checked when the eyes are fixed. In contrast with locomotor ataxy, this disease presents, as a rule, no ocular paralysis, no optic-nerve atrophy, no trophic changes, no visceral crises, and no affection of the sphincters. There is no mental change. As the disease progresses the muscles become weaker and more rigid, especially in the legs. The contraction of the calf-muscles leads to talipes equino-varus; and this is accompanied by extension of the toes, especially of the great toe, of which the proximal phalanx is extended, the distal flexed. Scoliosis is also often present.

The disease may last several years; death takes place generally from intercurrent affections, and not as a direct consequence of the spinal lesion.

Pathological Anatomy.—The spinal cord is often very small. The essential lesion is a sclerosis of the posterior and lateral columns,

FIG. 32

Cervical. Dorsal. Lumbar.

Sections of Spinal Cord from Case of Friedreich's Ataxia.
(*After Newton Pitt.*)

which is generally most intense in the lumbar region, and invades the whole of the posterior column. In the lateral column, the parts diseased are the pyramidal tract, the cerebellar tract, and the periphery of the cord in front of this. The pyramidal fibres in the anterior column are sometimes affected, and Clarke's posterior vesicular column has been found degenerated in association with the

cerebellar tract. The posterior nerve-roots are partially degenerated, but the peripheral nerves, as well as the anterior cornua of the cord, have hitherto been found free. Lissauer's tracts are usually intact.

The cause of the disease seems to be a congenital want of vitality in the neuron-systems, or tracts involved.

The **Diagnosis** of a case must depend upon the history of its association with other cases in the same family and its early appearance, the jerky unsteadiness of the head, the ataxy of the arms, the affection of articulation, and the nystagmus. By nearly all of these it is distinguished from ordinary *locomotor ataxy*. It may resemble, more or less, *ataxic paraplegia, disseminated sclerosis*, and *cerebellar disease*. In the first there is increased knee-jerk, and no nystagmus; in the second the movements are more oscillating, less jerky, and the speech of a peculiar "staccato" character not present in Friedreich's disease. In hereditary cerebellar ataxy, which resembles it closely, the onset is later, the knee-jerks are retained, trophic disturbances and spinal deformities are absent. Cerebellar tumours present the usual evidences of intracranial pressure, pain, sickness, and optic neuritis.

Prognosis.—This is unfavourable, the cases tending to become worse, though they may become stationary for some time.

Treatment is of little avail, but the methods suitable to tabes dorsalis may be tried.

PROGRESSIVE MUSCULAR ATROPHY

This is a chronic disease characterised by wasting of muscles, with weakness consequent thereon, resulting from degeneration of nerve-cells in the anterior cornua of the spinal cord. The names *wasting palsy* and *chronic anterior poliomyelitis* have also been used for it, but as it is not an inflammatory lesion, the latter is not suitable.

Ætiology.—Our knowledge of the origin of progressive muscular atrophy is very incomplete. The disease begins mostly in early adult life, between the ages of twenty-five and forty-five, and it is more common in males than in females. In some cases it appears to have its origin in muscular over-exertion; and it is also attributed to mental distress and to exposure.

Symptoms.—In the majority of cases (known as the Duchenne-Aran type) the disease begins in the upper extremities, and is seen first as a gradual atrophy of the short muscles of the thumb and little finger, so that the thenar and hypothenar eminences are flattened and disappear. The interossei muscles are also wasted, leading to depressions between the metacarpal bones; and when the atrophy is advanced, the shortening of the extensors, unresisted by the interossei, produces the peculiar claw-like deformity (*main en griffe*) in which the first phalanges are over-extended on the

metacarpals, and the middle and terminal phalanges are flexed on the first. Motor weakness accompanies, *pari passu*, the atrophy. These changes may occur in both hands, but often begin in one before the other, so that the hands are affected unequally. Other muscles are then involved; the deltoid is often the next to atrophy, but it may be the muscles of the upper arm, generally the biceps first; or of the forearm, when the extensors are affected before the flexors. The trapezius in its lower two-thirds, and the other scapular muscles, may be affected. The disease subsequently spreads to the trunk and neck, and the diaphragm and intercostals are sometimes involved, so as to cause serious difficulties in respiration. In the ordinary form of progressive muscular atrophy the legs are spared until very late; they may then be affected like the arms, or may be rigid, with or without wasting. This rigidity approximates the case to one of amyotrophic lateral sclerosis, and many writers consider the two complaints to be identical. The course of the disease is often exceedingly slow; it may be years before it spreads from the hands to the arms, and years again before other muscles are involved.

The electrical excitability of the muscles fails in proportion to the wasting. Usually faradic and galvanic excitability diminish together, but contractions can still be obtained, except when the wasting of any muscle is extreme. Then a partial form of reaction of degeneration may be found—*i.e.*, in the nerves, slightly diminished excitability to both currents; in the muscles, slightly diminished faradic excitability, and increased galvanic excitability, with slow contractions, and increase of ACC; or the reactions may fail altogether, that of the galvanic current lasting the longest.

The reflexes are commonly lost; the knee-jerk remains as long as the legs are unaffected. A constant feature of this disease is the occurrence of *fibrillary contractions*, which were at one time thought to be pathognomonic; it is now known that they occur in other atrophic conditions. They consist of slight momentary twitchings of a few fibres of the muscle, visible on the surface, painless, though perceptible to the patient, and recurring every two or three minutes. They occur spontaneously, but may be brought out by a tap on the skin over the muscle.

Sensation remains intact, and the bladder and rectum are normal.

Where progress is very slow and the limbs are the parts chiefly or alone affected, death only occurs from intercurrent diseases, such as phthisis, pneumonia, or bronchitis. But some cases are fatal through failure of the respiratory muscles, and in others the disease spreads upwards to the medulla oblongata, so that paralysis of the tongue, larynx, and pharynx results, constituting the *progressive bulbar paralysis*, which will be described shortly.

In another type of the disease, the atrophy first attacks the muscles of the legs, then those of the thighs and the glutei, but does not extend any higher.

In a third type (Duchenne's subacute ascending paralysis), the atrophy, which begins in the lower extremities, extends to the muscles of the back and trunk, and then invades the shoulders and arms, finally perhaps ending in a bulbar paralysis, as in the Duchenne-Aran type.

A fourth variety is the *peroneal form* of progressive muscular atrophy first described by Charcot and Marie, and by Tooth. This begins with atrophy of the peronei and anterior tibial muscles, and subsequently of other muscles of the foot and leg. It causes double talipes varus, and the limbs become cold and livid. Fibrillary tremors are sometimes, but not always, present; reaction of degeneration is mostly found. The atrophy may extend to the muscles of the arm and hand, producing then the *main en griffe*. It occurs in members of the same family, and generally begins early in life.

A very rare form is the *infantile hereditary progressive muscular atrophy* of Werdnig-Hoffmann. It begins in early infancy by involving the muscles of the hip girdle, then those of the trunk, and of the shoulder girdle; but never those supplied by cranial nerves. It lasts two or three years, and is fatal in less than four.

Anatomical Changes.—In the ordinary form of progressive muscular atrophy (Duchenne-Aran) changes are found in the anterior cornua of the spinal cord, in the anterior nerve-roots and nerve-trunks, and in the muscles themselves. The *anterior cornua* are scarcely, if at all, altered in size or shape, thus contrasting with the condition found in acute poliomyelitis; but they are pale, translucent, and almost entirely wanting in the large motor-cells or cell-bodies of the lower motor neurons. Such of these as remain are smaller than normal, globular in shape, and without processes. At the same time the connective tissue elements are increased. The posterior cornua are always normal. Sclerosis of the pyramidal tracts is present in those cases which present the features of amyotrophic lateral sclerosis (*see* p. 297); it is sometimes found, even when during life there have been no spastic phenomena. The anterior *nerve-roots* are visibly atrophied, being small and gray; but changes in the nerve-trunks are not so obvious, in consequence of the admixture of healthy fibres from the sensory roots. The *muscles* are pale and small. Under the microscope, the fibrillæ are found in different degrees degenerated. Some are simply diminished in size; in others, the striation is indistinct, or replaced by fatty granules; in others there is a marked longitudinal striation; and others again have undergone a vitreous or waxy change. The intervening connective tissue is increased in quantity.

The changes in the peroneal form of progressive muscular atrophy consist of sclerosis of the posterior columns, and posterior parts of the lateral columns, and degeneration of the cells of the anterior cornua, and of Clarke's columns. Atrophy of spinal motor cells and nerves is also found in the infantile hereditary type.

Diagnosis.—Progressive muscular atrophy has to be distinguished from all other diseases accompanied by atrophy of muscles, especially *primary muscular atrophy*, and *muscular dystrophy* (*see* Diseases of the Muscles). When the atrophy affects the hand alone, the deformity resembles somewhat the result of lesion of the *ulnar nerve;* but in this last the ulnar half of the hand is more decidedly affected (the radial lumbricales being supplied by the median nerve), and anæsthesia and trophic changes occur; in traumatic cases the history of injury will, of course, help. *Lead paralysis* is recognised by the extensors being first, and generally alone, affected; by the blue line on the gum, the detection of lead in the urine, and perhaps by the occupation, and preceding attacks of colic. *Multiple neuritis* is distinguished by the more rapid onset, the wide extent of the parts affected, the numbness or anæsthesia, and the tenderness of muscles. As contrasted with other diseases of the spinal cord, the important feature is the slow commencement by atrophy and weakness together, without pain, spasm, or sensory troubles. This distinguishes it from *tumour* and *meningitis*, which may cause muscular atrophy. In *acute poliomyelitis* of children and adults the history is completely different. In the typical cases of *amyotrophic lateral sclerosis* the course is more rapid and the reflexes are rapidly increased.

Prognosis.—This is unfavourable; but the progress of the atrophy is sometimes completely arrested.

Treatment.—Drugs have but little value; arsenic and strychnia have seemed to do good sometimes. General hygienic treatment should be pursued: good air, nutritious food, exercise without strain, and freedom from mental worry. In addition to this the muscles may be locally treated with electricity, massage, and passive movements; but the improvement to be obtained by these means is at best but very slight.

AMYOTROPHIC LATERAL SCLEROSIS

In this disease there is degeneration of the motor cells of the anterior cornua of the spinal cord, with sclerosis of the pyramidal tracts in the lateral columns. It thus combines at the same time the lesions of progressive muscular atrophy and of spastic paraplegia—that is, lesions of both upper and lower motor neurons. But the lesions are not confined to the spinal cord: the motor centres of the bulb (especially the hypoglossus and vago-accessory nuclei) are also affected towards the end of the disease in the majority of cases; and the atrophy of the upper neurons in prolonged cases extends through the medulla oblongata, crura cerebri, and internal capsules to the motor cells in the cortex of the brain. Atrophy and sclerosis also affect some of the association fibres of the antero-lateral columns near the gray cornua. It is held by some that progressive muscular atrophy and amyotrophic lateral sclerosis are the same disease.

Ætiology.—It occurs between the ages of twenty-five and fifty, is more frequent in females than in males (Charcot), but cannot generally be referred to any particular cause.

Symptoms.—The first symptom is weakness in the upper extremities, which are soon seen to be affected with wasting. This is not limited to the interossei, or other muscles of the hand, though it may begin in them, but affects the whole upper extremity much more equally (according to Charcot) than in progressive muscular atrophy. Fibrillary contractions often occur, and the electrical reactions, as in progressive muscular atrophy, show only a simple diminution, unless the wasting is extreme, when reaction of degeneration may be present. Quite early in the history the tendon jerks are increased, and can be elicited on striking the tendons of the biceps and triceps, or the lower ends of the radius and ulna. After a time rigidity takes place in the atrophied muscles, and considerable contractures may result. Charcot especially noted a deformity which he regarded as characteristic of amyotrophic lateral sclerosis: the upper arm lies close along the body, the forearm is semiflexed and pronated, whilst the wrist is strongly flexed, and the fingers are bent into the palm. Generally, after eight to twelve months, the lower extremities become involved, presenting at first the characteristics of spastic paraplegia. Weakness and rigidity appear together, the former being masked by the latter. The knee-jerk is increased, and ankle-clonus can be obtained; the electrical excitability remains; and walking can be accomplished, though with difficulty. After some time, wasting also occurs in the lower extremities, but it is never so complete as in the upper. Sensation and the sphincters are unaffected. As the disease spreads to the bulb, the tongue, lips, palate, and laryngeal muscles are paralysed; deglutition and speech are rendered difficult, and the characteristic features of palatal paralysis or laryngeal paralysis may be present. The facial muscles are also atrophied, and in later stages rigidity ensues, with increased jaw-jerk or masseter-clonus. With the extension to the cerebrum the emotional faculties are disturbed, and the patients laugh or cry without good cause.

The duration is from one to four or more years, and is shortest in those cases in which the bulbar symptoms develop early. Death results from asphyxia, inanition, or more often from pneumonia, caused by inhalation of food particles through the larynx.

Diagnosis.—*Progressive muscular atrophy* (Duchenne-Aran type) has a slower course, with no excess of tendon reflexes or muscular rigidity. *Primary spastic paraplegia* begins generally in the lower extremities, and is unaccompanied by atrophy.

Treatment may be tried on the same lines as in these two diseases, but the prognosis is very unfavourable.

DISSEMINATED SCLEROSIS

(*Multiple Sclerosis, Insular Sclerosis, Sclérose en Plaques Disseminées*)

This disease is characterised by the development of numerous patches of chronic inflammation or sclerosis throughout the central nervous system. It has been described as occurring in three forms—cerebral, spinal, and cerebro-spinal; but the last is by far the most frequent. At an autopsy of such a case the surface of the spinal cord, medulla oblongata, pons varolii, and the base of the brain presents a number of irregular patches of pinkish-gray colour, rather sharply outlined, and contrasting with the natural white colour of the medulla, pons, and crura. On section, the discoloration is found to extend inwards so as to form deposits of a round or oval shape, ranging in size from that of a pea to that of a hazel-nut, generally harder than the normal nervous tissue, and even leathery, or cartilaginous; sometimes projecting above the level of the section, sometimes sunken below it. Recent patches are dark gray, older patches more yellowish-gray, and less translucent. They affect the white matter more than the gray matter; thus in the spinal cord the greater part of the cornua is unaffected, and in the cerebrum they are best seen on section of the hemispheres, which are dotted with the gray areas, and the walls of the lateral ventricles are often invaded. They are not frequent in the cerebellum; but the sclerosis may invade the olfactory bulbs, and the spinal and cranial nerve-roots. Under the microscope the outline of the patch or nodule is much less distinctly marked than it appears to the naked eye. The nodule consists chiefly of fibrous or finely fibrillated tissue, developed by overgrowth of the neuroglia; within this area the nerve fibres have lost their myelin sheaths, but great numbers of axis-cylinders persist. Nerve cells are very little involved unless late in the disease. There may be some thickening of vessels, but it is rarely pronounced. It is remarkable that secondary degeneration is quite rare.

Ætiology.—The disease has no marked preference for one sex over the other. It occurs mostly in youth or middle age, and cases are recorded of the characteristic symptoms even in children. Many cases have now been recorded as following upon different acute infectious diseases, such as typhoid fever, malaria, influenza, pneumonia, scarlet fever; and chronic metallic poisoning, as *e.g.*, by tin, has been known as an antecedent. Cold, mental worry or excitement, and injuries are still credited with being causes. But in many cases there is no obvious antecedent, or it has occurred long before the first symptom.

Symptoms.—The onset of the disease is very variable; sometimes it is quite gradual, and the patients simply notice that they get weaker in the legs, or nervous, or tremulous or spastic. In other cases there has been an apparently more rapid beginning—the knees have suddenly given way, or there has been sudden weakness of one arm or leg; this has perhaps recovered after a

time, and then the same or another limb has become paretic, so that it is very easy for the early symptoms to be regarded as hysterical. In other cases, amblyopia, temporary or more persistent, with or without optic atrophy, may be the first symptoms: in others, again, numbness and anæsthesia. The recurrence of these symptoms, or the persistence of some of them in a slight form, may go on for years; but ultimately in most cases the following three symptoms are present in more or less pronounced degree, and in some instances they may develop even early. They are: (1) *Tremor of muscles on attempting to move;* (2) a peculiar manner of speaking, *syllabic* or *scanning speech;* and (3) *nystagmus.*

The tremor is of that kind described as *intention-tremor* (*see* p. 200). It is best observed in the hand and arm when the patient attempts to take hold of an object—the limb oscillates irregularly to the right and left, or up and down, with regard to the object aimed at, the excursions from the straight line being often several inches in extent. When the patient sits up in bed, or stands up, the body swings to and fro, and the head undergoes a series of nodding movements; while in the attempt to walk the movements of the legs are similarly unsteady. When the patient is lying quiet in bed, or sitting with the back, head, and arms supported, he is perfectly still. The movements are more violent the greater the effort, and the more the patient feels that he is under the observation of others.

In talking, every syllable is distinctly uttered in a slow, deliberate manner and somewhat suddenly, as in the *staccato* delivery of music. There is little or none of the natural slurring of some syllables and accentuation of others. The voice is also rather high-pitched and monotonous. It has been observed with the laryngoscope that the vocal cords are apt to relax in phonation, and to undergo rapid changes of tension.

The *nystagmus,* or oscillation of the eyeballs, is, like the movement in the limbs, only brought on by voluntary movements—that is, when the eyes are fixed upon the object, or when they are much turned to one side. It is thus unlike the oscillation seen in some cerebellar tumours, or the constant movement which occurs in some diseases of the fundus of the eye. Paralysis of ocular muscles, especially of the external recti, sometimes occurs.

The motor power is generally diminished, and the knee-jerks are exaggerated; and sooner or later rigidity of the lower extremities occurs, when ankle-clonus and Babinski's sign will also be observed. The rigidity is pronounced in some advanced cases, where it may co-exist with actual paralysis, from the sclerosis invading nearly the whole thickness of the cord at one spot, and thus, like a transverse myelitis, producing paraplegia. But in earlier stages rigidity with extension and adduction of the legs may be a troublesome symptom, and the gait will have a *spastic* character. Occasionally, atrophy of muscles is observed, when it may be supposed that sclerosis has invaded the gray cornua; and ataxy is sometimes seen. Mostly

electric irritability is normal; only in later stages it may be diminished, or the reaction of degeneration may be observed, where muscular atrophy is present.

There may be modification of sensation, such as numbness or formication, and sometimes pains, but rarely complete anæsthesia.

The bladder, rectum, and sexual organs often retain their power, or there may be some impairment of the functions of the bladder—involuntary expulsion or slight retention, perhaps temporary and relapsing. Rectal incontinence is uncommon.

There is sometimes defect of vision, with diminution of the field and achromatopia, or central scotoma, and rarely complete blindness. This is often associated with pallor of the disc or marked but partial optic atrophy; but there may be defects of vision without obvious change in the disc, or pallor of the disc without loss of sight. The atrophy is not generally preceded by neuritis, and rarely becomes complete. Headache and vertigo are occasionally present. More frequent is some impairment of the mental or emotional powers. The patients are especially liable to laugh or cry without apparent reason, and the intelligence is weakened as the disease progresses. Delusions of grandeur may occur, and the case may ultimately assume all the characteristics of general paralysis of the insane. In some instances peculiar apoplectic attacks occur. The patient is seized with headache, giddiness, then loss of consciousness, and weakness of the arm and leg on one side. The face is red, the pulse is frequent, and the temperature rises to 104° to 106°. Recovery takes place in a day or two. Epileptiform attacks are more rare. Occasionally the usual symptoms may be absent, and death takes place from an apoplectic attack; and in other cases from special localisation of the patches, hemiplegia, or one of the various chronic spinal diseases, myelitis, spastic or ataxic paraplegia, amyotrophic lateral sclerosis, or bulbar paralysis may be simulated.

The disease may last several years—ten, fifteen, or twenty; but in a few cases improvement or even recovery has occurred. Death may take place much earlier from an apoplectic seizure, or the patient may be bed-ridden for years with paraplegia, and die from the accidents associated with that condition, or from intercurrent disease.

Pathology.—The symptoms are not at present satisfactorily explained. The tremors have been referred (1) to the want of "insulation" of the axis-cylinders in the sclerosed patches; (2) to the localisation of patches in the pons varolii or higher parts of the motor tracts; and (3) to the unequal innervation of the muscles intended to be moved, and their antagonists.

A possible explanation of the disease is that toxins carried to them by the blood-vessels cause degeneration of the myelin-sheaths, and that this is followed by neuroglial overgrowth, or sclerosis.

Diagnosis.—This is easy when the three cardinal symptoms are alone prominent. The tremors can scarcely be confounded with

chorea or paralysis agitans. In *chorea* the movements are more twisting, jerking, or writhing, and occur during rest. In *paralysis agitans* they are regular and rhythmical, more rapid, and less extensive; they occur during rest, and may, in early stages, be stopped by voluntary effort. The oscillating movements which are seen in disease of the *cerebellum* closely resemble those of sclerosis, but the former may be distinguished by the other evidences of local disease. It may be, as already implied, that they have the same origin. Charcot said that *chronic cervical meningitis* with cortical sclerosis might produce similar tremors. *Mercurial poisoning* resembles disseminated sclerosis in the occurrence of intention-tremors of the limbs, but the head and trunk are less affected in the former. In particular cases there may be some difficulty in distinguishing the disease from the spinal complaints already described, such as spastic paraplegia or a combined sclerosis, especially if, as is sometimes the case, the tremor is absent. The occurrence of attacks of temporary paralysis, or amblyopia, or numbness in a young person is very likely to be taken for *hysteria*. A hasty judgment must not be formed, but the case should be watched for other spinal symptoms. Nystagmus, changes in the optic disc, and incontinence of urine or fæces indicate an organic lesion.

The **Prognosis** is unfavourable, and the **Treatment** must be conducted on similar lines to that of other chronic spinal diseases. Arsenic, potassium iodide, mercury, quinine, may be tried. Rest in early stages is of great importance, and may be supplemented by massage, and the constant galvanic current.

SPINAL MENINGITIS

The spinal membranes may be affected separately, but it is common for inflammation beginning in one to spread to the other. Inflammation of the pia mater is called *leptomeningitis*, that of the dura mater *pachymeningitis*. The forms of inflammation commonly recognised are—first, an inflammation of the outer surface of the dura mater, usually set up by disease in the neighbourhood—this is called *meningitis externa* or *pachymeningitis externa;* secondly, inflammation of the internal surface of the dura mater, generally chronic and resulting in great thickening of the membranes (*pachymeningitis interna hypertrophica*), or in extravasation of blood, and the formation of fibrinous, pigmented deposits (*pachymeningitis interna hæmorrhagica*); lastly inflammation, beginning in the pia mater, or *leptomeningitis*, which may be acute or chronic. It will be seen that the symptoms of all these forms present much similarity, as they depend not so much on any alteration in the membrane itself as upon the implication of the nerve-roots which pass through them, and of the cord which they enclose.

ACUTE LEPTOMENINGITIS—ACUTE SPINAL MENINGITIS

Ætiology.—Its causes are often obscure, but amongst those to which it can be traced are exposure to injuries, such as fractures and dislocation of the spine, and various forms of infection. Thus it has sometimes arisen in connection with pneumonia, scarlatina, typhoid, septicæmia, or the puerperal state. A tubercular spinal meningitis not infrequently accompanies tubercular meningitis of the brain; and the two membranes are associated together in the disease known as epidemic cerebro-spinal meningitis or cerebro-spinal fever (*see* p. 88). Inflammation may spread from outside the spinal canal, first causing an external meningitis, or from the cerebral to the spinal membranes. It has also followed puncture of a spina bifida, doubtless from local infection.

Symptoms.—An attack of acute spinal meningitis usually begins with rigor and elevation of temperature; there is severe pain in the back, which may be localised, or extend along the whole length, and this pain is usually increased by movement as well as by pressure. In addition, there are paroxysmal pains of shooting, darting character, radiating in the course of the nerves arising from the part; and hyperæsthesia of the skin, even of the muscles, in the same areas. Irritation of the anterior nerve-roots leads to spasm of the muscles, producing rigidity of the spine, with more or less severe arching, or *opisthotonus*. This may be present in the whole length of the spine, or affect the neck alone, when the occiput is fixed back between the shoulders (retraction of the head, cervical opisthotonus). The abdominal muscles and the muscles of the legs are also affected by spasm, and the spasms are increased by attempts at movement. The reflexes are generally increased, and Kernig's sign may be observed. The bowels are constipated, and the urine is retained by spasm of the sphincter, or later by paralysis of the detrusor. There are the usual accompaniments of pyrexia; the temperature is generally raised, the pulse is quick, and the "tache méningitique" (*see* p. 356) is well marked. After some time—it may be a few days or longer—the symptoms of irritation, pain, and spasm give place to those of paralysis, anæsthesia, and diminished reflexes; and the case then approximates to one of myelitis, and is either rapidly fatal from exhaustion, paralysis of the respiratory muscles, or acute bed-sores; or lapses into a more chronic condition, with atrophy and shortening of muscles. Some patients recover after several months; others die from bed-sores, or vesical, renal, or other visceral complications.

The symptoms of meningitis vary considerably, and are not always so manifest as above indicated. The signs of irritation may be of very short duration, and those of paralysis become prominent quite early; and in cases where the cerebral membranes are involved at the same time, the spinal symptoms may be entirely

masked, as is frequently the case in tubercular meningitis. In cerebro-spinal fever the symptoms of the two conditions are combined. Further, differences are observable according as the inflammation affects mainly the cervical, the dorsal, or the lumbar region. It is sufficient here to point out that, in cervical meningitis, retraction of the head, dyspnœa from implication of the diaphragm, dysphagia, inequality of the pupils, and great elevation of temperature are likely to occur; while in dorsal meningitis the trunk muscles, and in lumbar meningitis the muscles of the loins and legs, are especially affected. The duration varies from two or three days to three weeks in the more acute cases, and is occasionally much longer.

Morbid Anatomy.—In the early stage the pia mater is reddened from increased vascularity, and small hæmorrhages may be present; an exudation then takes place in the meshes of the pia mater and upon the surface, at first gray in colour, subsequently purulent and yellow or greenish-yellow. This deposit is seen often over a large extent of the cord in irregular patches, more upon the posterior than the anterior aspect from the effects of gravitation. The spinal fluid is turbid and opaque. The inflammation affects also the inner surface of the dura mater, and the two membranes may be united by the viscid purulent lymph. The process frequently extends into the cord (*meningo-myelitis*), affecting especially its periphery, in which dilated vessels and extravasated leucocytes occur, and the nerve-roots are similarly involved. Bacteria have been found in some cases, especially pyococci, diplococcus pneumoniæ, and a diplococcus, allied to the diplococcus intracellularis of Weichselbaum (Risien Russell).

Diagnosis.—Meningitis has mainly to be distinguished from myelitis and from tetanus. As contrasted with meningitis, there is less fever in *myelitis*, the local and radiating pain, rigidity, and spasms are less marked or absent, and paralysis and anæsthesia come on quite early. *Tetanus* is distinguished by the persistent spasm, aggravated on the slightest peripheral irritation, by its commencement in trismus, by a slight pyrexia, if any, and by the history of wound in the majority of cases. Rheumatism of the lumbar muscles or of the vertebral articulations may to a certain extent simulate meningitis. The diagnosis both of the existence and nature of meningitis has now often been settled by means of the operation of *lumbar puncture*, followed by microscopical and bacteriological examinations of the fluid withdrawn (*see* p. 213).

Treatment.—In the acute stage the treatment is similar to that of acute myelitis, with this addition—that perfect rest and avoidance of all external irritations should be ensured; while morphia, chloral, and potassium bromide, or chloroform inhalations should be given to relieve pain. Mercury and potassium iodide may be used internally, or the oleate of mercury may be rubbed into the spine. In the less acute stages, mercurials and potassium iodide are to be given internally, and counter-irritants may be applied locally.

CHRONIC LEPTOMENINGITIS

Ætiology.—This may be only the continuation of an acute process; or it may be chronic from the first, and is then attributable to syphilis, alcoholism, or injuries. It is commonly found in association with inflammatory (or degenerative) processes in the cord itself, such as chronic myelitis and posterior sclerosis; and it may also result from lesions external to the cord.

Anatomy.—The condition is one of fibrous thickening of the pia mater, with dilatation and thickening of the walls of the blood-vessels: in syphilitic cases the characteristic gummatous deposits or gelatinous exudations are present. The change may be extensive when it follows upon an acute lesion, more limited and scattered when it is chronic from the first. The parts affected are in some cases determined by the pre-existing lesion of the cord. The pia mater and arachnoid may be adherent to the dura mater; the cord itself often shows inflammatory changes in the connective tissue at the surface (annular myelitis), or a more extensive myelitis; and the nerve-roots may be compressed and atrophied.

Symptoms.—These are the same in kind as those of acute meningitis—namely, symptoms due to local irritation of the membranes, others due to implication of the nerve-roots, and others again which result from implication of the cord; but they are gradual in their onset, they are not accompanied by fever, the muscular spasms and rigidity are much less marked than in the acute form, the sensory symptoms are often more localised, and paralysis may occur early. Ultimately the case may assume all the features of chronic myelitis.

Treatment.—Mercury and potassium iodide should be freely used; and help may be derived from massage, warm baths, and spinal douches.

INTERNAL PACHYMENINGITIS

It has been already stated that the inner surface of the dura mater is often involved in both acute and chronic leptomeningitis; but there are two forms of pachymeningitis which require separate mention—namely, *pachymeningitis interna hypertrophica* and *pachymeningitis interna hæmorrhagica.* In the former, which is probably often due to syphilis, the dura mater becomes immensely thickened, so as to cause very severe compression of the cord and nerve-roots.

Symptoms.—Charcot has described it as especially affecting the cervical region (hypertrophic cervical pachymeningitis). After a first stage of shooting pains in the back of the neck, shoulder, arms, and upper part of the thorax, with muscular twitchings and spasms, there are gradually developed anæsthesia, paralysis, and atrophy, and loss of electrical reactions in the distribution of the compressed nerve-roots. Charcot observed that the median and

ulnar nerves were more affected than the musculo-spiral, and that consequently there arose a deformity of the upper limb, characterised by extension at the wrist, and metacarpo-phalangeal joints, and by flexion of the phalanges. This appears to be due to implication of the lower part of the cervical region. In a case affecting the upper part of the cervical region the muscles supplied by the musculo-spiral were paralysed, and a condition of extreme flexion was the result. As the compression of the cord increases, paraplegia, secondary degenerations, and spastic rigidity of the paralysed parts occur. In rare cases, hypertrophic pachymeningitis affects the lumbar region, or the cauda equina, when the pains, paralysis, and atrophy are situate in the lower extremities, and the sphincters are involved early (*see* p. 314).

Treatment.—A vigorous antisyphilitic treatment should be employed.

In *pachymeningitis interna hæmorrhagica*, or *hæmatoma of the dura mater*, the inner surface of the membrane is covered with a reddish-brown exudation, consisting of fibrin, connective tissue, recent extravasations of blood, and, it may be, cysts containing blood in different stages of change. This form of meningitis affects the greater part of the cord, and is associated mostly with a similar condition in the cerebral dura mater (*see* p. 362). The disease has been attributed to the abuse of alcohol, and it is seen in some cases of mental disorder.

The **Symptoms** are those of a slight chronic meningitis, but they are often masked by others which result from the cerebral lesion.

EXTERNAL PACHYMENINGITIS

As already stated, this is mostly secondary, and arises from any inflammation in the neighbourhood of the dura mater: thus, deep bed-sores over the sacrum may slough into the spinal canal, or the dura mater may be inflamed by caries of the spine (the most common cause), by retro-pharyngeal abscess, or abscess in other situations, by cellulitis of the neck, or inflammation of the subpleural tissue; or it may result more directly from injury. It may be acute or chronic. In the acute form, such as occurs from sloughing bed-sores, the external surface of the dura mater is reddened, or presents lymph upon the surface, or may be covered with a layer of pus. In caries of the spine the process is generally more chronic, and the outer surface of the membrane is covered with caseous or semi-caseous deposit, and the membrane is itself thickened. More or less inflammation or compression of the nerve-roots commonly co-exists, and the cord may be much narrowed. In the purulent cases especially the process may extend to the external surface.

Symptoms.—These are not essentially different from those already described under leptomeningitis. They are pain in the back of the seat of the lesion, stiffness of the back from rigidity of the muscles, and pain on movement, pains in parts corresponding

to the nerves arising from the region affected, hyperæsthesia, jerking and tension of the muscles, and later, the symptoms of compression of the cord—namely, paralysis, and anæsthesia, in varying degrees, of the parts below the seat of the lesion. In acute cases the symptoms of irritation are likely to predominate; in chronic cases those of compression.

Diagnosis.—The important point is the recognition of an external cause, otherwise it may be impossible to distinguish it from other forms of spinal meningitis.

The **Prognosis** is unfavourable in acute cases, but more hopeful in caries of the spine, of which a large number of cases make a more or less perfect recovery.

Treatment.—The chief indication is to remove the original cause, if possible; the others, to aid in the absorption of inflammatory products, and to treat the myelitis resulting from compression.

SPINAL MENINGEAL HÆMORRHAGE

This is a rare occurrence. It arises from injuries, such as blows, stabs, falls on the feet or back, and traction on the spine of newborn children during delivery. Blood effused into the cranial cavity sometimes runs down into the spinal canal; and very rarely, an aneurysm of the aorta or of the vertebral artery has burst into it. Hæmorrhage into the membranes may form part of purpura and scurvy, or of other general conditions in which hæmorrhage occurs, such as alcoholism; and in this connection it has already been mentioned as part of pachymeningitis hæmorrhagica.

Symptoms.—Like those of hæmorrhage into the cord itself, the symptoms are distinguished by the suddenness of their onset. There are severe pains in the back, pains radiating along the nerves, and spasms and rigidity of the muscles supplied by the nerves. Subsequently there is loss of power and sensation, and in some cases complete paralysis and anæsthesia occur. The paralytic symptoms follow quickly upon the signs of irritation, and reach their height in a period varying from a few hours to a few days. Death, also, may happen in a few hours, or symptoms characteristic of meningitis may supervene.

Diagnosis.—Spinal meningeal hæmorrhage is distinguished from *hæmorrhage into the cord* chiefly by the fact that signs of irritation, such as muscular cramps, spasms, rigidity and hyperæsthesia, precede paralysis, and the paralysis is less complete. *Spinal meningitis* is more gradual in its onset, and is accompanied by fever from the first.

Prognosis.—This is sufficiently serious, and many cases are fatal. On the other hand, recovery is more frequent than in hæmorrhage into the cord.

Treatment.—This must be conducted in the same way as that of intra-medullary hæmorrhage. The later treatment is that of spinal meningitis.

TUMOURS OF THE SPINAL CORD AND ITS MEMBRANES

Tumours may grow in the spinal cord, in the spinal membranes, or from parts of the spinal canal outside the membranes. Of these latter (*extra-dural*) growths, the most common are tumours growing from the vertebræ, such as sarcoma and carcinoma; but the inflammatory material resulting from caries of the spine also forms masses which act like tumours in compressing the cord. Fatty tumours and hydatid cysts occur outside the membranes, but are rare. Within the membranes (*intra-dural*) the following growths occur: sarcoma, glioma, tubercular tumours, gumma, myxoma, lipoma, myo-lipoma, neuroma, fibroma, hydatid cysts, and cysticercus telæ cellulosæ. The first four are the most common. Some of these tumours grow from *nerve-roots*, especially sarcoma, myxoma, fibroma, and glioma. The tumours arising within the *cord* itself are especially sarcoma, tubercle, glioma, and gumma (*see* Tumours of the Brain).

The tumours are generally isolated and of small size. There may be two or more tubercle-masses, syphilitic gummata are occasionally multiple, and there may also be several neuromata at the same time. Tumours of the cauda equina may attain a considerable size from the greater space allowed them. A glioma forming around the central canal (peri-ependymal glioma) and extending a variable distance along the cord may by its softening lead to the formation of an elongated cavity or *syringomyelia* (*see* p. 309).

In their continued growth the tumours produce important effects upon the cord and nerves. Extra-medullary tumours cause compression and wasting of the nerve-roots, or press upon the cord and set up myelitis and subsequent secondary degenerations. Similarly, myelitis and secondary degenerations may follow the growth of tumours in the cord itself. Occasionally the vessels of a glioma rupture, and the symptoms of a spinal hæmorrhage develop with their usual suddenness.

Ætiology.—The causation of spinal tumours cannot be treated of apart from that of tumours in general. It is doubtful whether some cases have been due to blows or injuries. Tubercle and gumma occur as parts of a general morbid condition. Some tumours are probably congenital (*e.g.*, myo-lipoma).

Symptoms.—These symptoms of a spinal tumour vary with its situation.

Within the cord, at first gradually displacing parts, it will at length produce sufficient compression to interrupt conduction, and paralysis and anæsthesia below the lesion will slowly develop. The paralysis often precedes the loss of sensation. If the tumour is limited to one side, the symptoms will be unilateral, with paralysis of one side and anæsthesia of the other. If it involves gray matter to any extent, there will be atrophy of the nerves and muscles proceeding from the part. Ultimately the tumour, by continued

growth only, or by setting up myelitis, is likely to cause complete paralysis, with secondary degenerations, and increased reflexes and rigidity of the muscles of the lower extremities. If the tumour is situated in the lumbar enlargement of the cord, the muscles are atrophied, the reflexes are lost, the electrical reactions are altered, and the sphincters are implicated.

Tumours outside the cord give rise to symptoms indicating irritation or compression of the nerve-roots, especially severe local pain in the spine, sharp pains shooting from the same spot in the direction of spinal nerves, rigidity of spinal muscles, hyperæsthesia or localised anæsthesia, and muscular weakness. These symptoms may be unilateral, and limited to one or other region of the spinal cord—cervical, dorsal, or lumbar. After some time—it may be months or a year or two—symptoms of compression of the cord or local myelitis appear, consisting of paralysis and anæsthesia below the seat of the lesion, with spastic rigidity, increased reflexes, and vesical troubles, and perhaps herpes zoster and trophic changes. If the tumour is unilateral, the symptoms will for a time at least be such as are caused by a unilateral transverse lesion, namely, pain and paralysis on one side and anæsthesia on the opposite side (*see* p. 262). In multiple tumours the symptoms may be more varied; in a case already mentioned (*see* p. 285) tabes dorsalis was closely simulated. Ultimately in nearly all cases the termination is fatal, in the same way as in myelitis—either through failure of the respiratory muscles, or through bed-sores and exhaustion or pyæmia, or through cystitis and renal complications. (*See* also Compression of the Spinal Cord.)

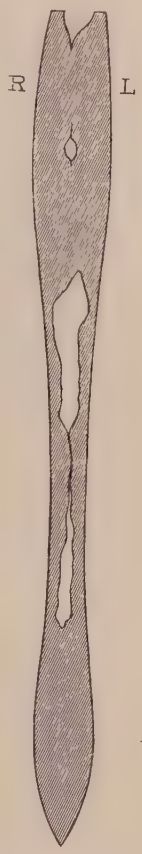
Diagnosis.—This can, of course, be only made after a certain time has elapsed. The distinctive features are severe continuous pain and other symptoms of irritation, limited to one or two segments of the cord, followed by progressive paralysis, without any evidence of caries or injury to the spine. Tumours in the cord itself may cause paralysis, without preceding pain or spasm, and the symptoms are early bilateral; if the tumour grows in the membranes, the symptoms may be limited to one side for a long time. The diagnosis from *myelitis* depends chiefly on the slow development of the symptoms and their reference to a strictly limited spot. The nature of the tumour may be difficult to determine: a syphilitic history and the association of cerebral symptoms indicate gumma; and preceding cancer may give a clue in some cases. Tubercle occurs in early life, and may be suggested by a strong family history, or by indications in the patient; but a tubercle of the cord is infinitely rare even amongst phthisical persons, and hence too much stress should not be laid upon this point.

Prognosis is very unfavourable. Gummata may yield again and again, or entirely, to treatment, and tubercles may possibly in a few cases become obsolete; but in the majority of instances the disease can only terminate fatally in the manner above indicated.

Treatment.—Where there are good grounds for diagnosis of

gumma, vigorous anti-syphilitic treatment should be employed; that is, full doses of potassium iodide, and mercurials—either the perchloride internally, or the ointment or oleate rubbed into the skin. Even when the syphilitic nature is uncertain the same treatment may be for a time tried, since other forms of tumour are not amenable to any drugs, with the exception of tubercle-masses, in which case the small chance of recovery may be improved by general tonics, iron, cod-liver oil, and similar remedies. Failing these measures, the help of surgery should be considered. Operations have been successful in some cases, and are more likely to be so when the tumour is not actually within the cord itself. Symptomatic treatment will generally be required, as in other instances of paraplegia; bed-sores and vesical lesions must be guarded against, pain alleviated, &c.

FIG. 33



Spinal Cord showing the Cavity in Syringomyelia.

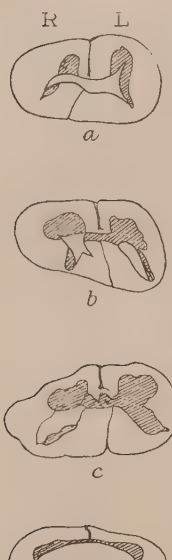
SYRINGOMYELIA

The elongated cavities occasionally found in the spinal cord may be a simple slight deviation of the central canal of the cord (*hydromyelia*), or a more definitely pathological condition called syringomyelia (σῦριγξ, a pipe). In the latter there is usually a single cavity, occupying the cervical and upper dorsal regions, lying in the posterior half of the cord behind the commissure, or in one or other posterior cornu. It is elongated, several inches in vertical extent, and is very variable in diameter at different levels of the cord. On transverse section it may have the appearance of a fissure running transversely from side to side, or may form a circular or oval space, occupying the greater part of the posterior half of the cord. At one or more points in its length it may be divided into two, thus forming a cavity in each posterior cornu. The cavity is often bounded by a thin layer of dense fibroid tissue, and surrounded by tissue which is translucent, gelatinous, deficient in nerve-fibres or other structural elements, and consists of neuroglial tissue of an embryonal character. In some cases there is a growth of sarcoma or glioma in the tissue around the cavity. The cavity is sometimes partly lined with epithelium like that of the central canal, and sometimes, no doubt, arises from it.

The condition is often congenital; and the cavities have been found in young children in association with hydrocephalus, or distension of the cerebral ventricles. They may then be due to

the inadequate filling-up of the posterior portion of the cavity which is formed from the primitive groove; and around this cavity the embryonal tissue persists. In later life there is both an increase of neuroglial or gliomatous tissue, and the cavity is enlarged. In some cases it is believed that the primary change in adult life is a gliomatous growth, and that this breaks down to form a cavity; in others that a hæmorrhage has been the original lesion.

FIG. 34



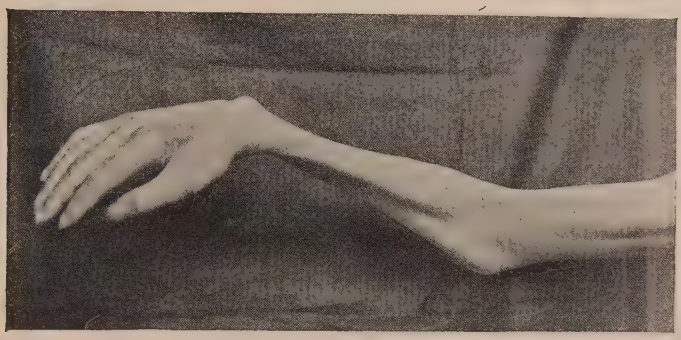
Tranverse Sections of Spinal Cord affected with Syringomyelia. *a*, *b*, *c*, *d*, cervical region; *e*, dorsal region; *f*, lumbar region. The asymmetry is produced in the course of preparation.

Symptoms.—The condition is not always accompanied by symptoms, and it has often been discovered *post mortem* when not suspected. In other cases there has been a great variety of symptoms, which have come on insidiously, and generally first in early adult life. The most constant results are as follows. There is loss of sensation to pain and variations of temperature, while sensation to touch and the muscular sense are unaltered (*dissociation*); as a result of this patients may suffer burns and injuries which they would escape under normal conditions. Muscular atrophy, progressive in its development, affects the upper extremities especially. The small muscles of the hands are wasted, producing the *main en griffe*, and subsequently the forearms and higher parts are involved, giving an appearance like that of progressive muscular atrophy (*see* p. 293): there may be fibrillary contractions, and the reflexes are diminished. The legs are less often implicated, but they may be weak or become slightly spastic. A third feature of interest is the occurrence of trophic disturbances, such as œdema of the fingers, local disturbances of sweating, bullæ, ulcers, whitlows (*Morvan's disease*), loss of the nails, and hypertrophy, atrophy, or brittleness of the bones. The joints, especially the shoulder and elbow, may present changes very like those described under tabes dorsalis; the ligaments and capsule become relaxed, and the head of the bone is absorbed. Spinal curvature (kyphosis, lordosis, and skoliosis), and contraction of the palpebral fissure, with retraction of the eyeball and small pupil, also occur. The bladder and rectum escape as a rule. Rarely, the lesions spread to the medulla and pons so as to produce paralysis of some of the muscles supplied by the cranial nerves. The disease may undergo

spontaneous arrest, and the patients often live for years; they die eventually from complications, such as cystitis and bed-sores.

The muscular paralysis and atrophic conditions are explained by the growth in or pressure upon the central gray matter. The dissociated anæsthesia is in accordance with the belief that the

FIG. 35



Illustrates Muscular Atrophy in a Case of Syringomyelia.
(*After Turner and Stewart.*)

sensations of pain and temperature are conveyed by fibres which pass into the gray matter, there decussating, and probably ascending by the antero-lateral tract of Gowers (Van Gehuchten). Thus the dissociated anæsthesia has a vertical extent corresponding to that of the cavity in the cord, and is accordingly limited to the upper half of the body in some cases.

Diagnosis.—In the early stages the disease is likely to be mistaken for multiple neuritis or progressive muscular atrophy. In the former the legs are more decidedly paralysed; in the latter there are no sensory troubles. Difficulties have also occurred with leprosy and with Raynaud's disease. In the advanced cases the dissociated anæsthesia, the muscular atrophy and paralysis, and the trophic disturbances of the skin are distinctive.

Treatment.—This can scarcely be much more than symptomatic. Arsenic internally may be tried: and recently the application of Roentgen rays to the spine has appeared to be beneficial.

COMPRESSION OF THE SPINAL CORD

It is desirable to deal separately with compression of the cord, although it has been frequently alluded to in the description of myelitis, meningitis, and tumours. The most common cause of compression of the cord is undoubtedly caries of the spine, not from "angular curvature," which the caries produces, but from the inflammatory or caseous products, which form between the diseased

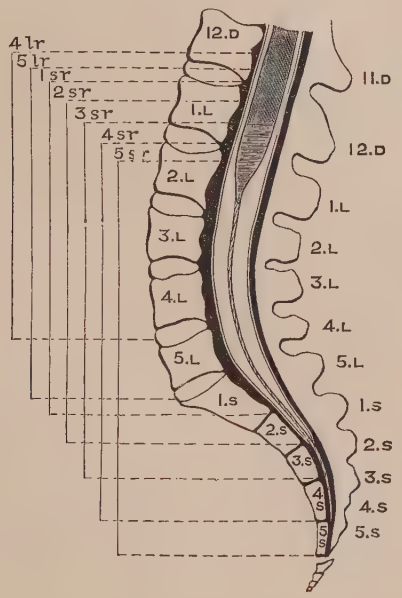
bone and the external surface of the dura mater, destroying the posterior common ligament and setting up an external pachymeningitis. The other less frequent causes are tumours, growing from the bones or membranes, aneurysm eroding the spinal column, and chronic thickening of the membranes (hypertrophic meningitis).

Whatever the cause of the compression is, the cord is narrowed, it may be to one-half or one-third its diameter, and myelitis is set up in the compressed part and in the cord immediately adjacent. Subsequently degeneration takes place in the posterior columns above the lesion, and in the pyramidal tracts below the lesion. To the naked eye and the microscope the changes are like those which are seen in myelitis from other causes—destruction of nerve-elements and increase of connective tissue; but even after prolonged and considerable compression it is remarkable that many nerve-fibres may remain intact.

Symptoms.—These are determined partly by pressure upon the nerve-roots, and partly by pressure upon the cord itself. The former occur first, and consist of pains of burning or neuralgic character in the area of the nerves whose roots are compressed. They differ from true neuralgia, according to Charcot, in not possessing *points douloureux*. Accompanying them is hyperæsthesia, at points corresponding to the distribution of the nerves; and later on anæsthesia supervenes, and may coexist with the severe pains, constituting the so-called *anæsthesia dolorosa*. Occasionally, trophic disorders of the skin are present, such as zona, bullæ, or eschars. Compression of the anterior nerve-roots may lead to atrophy of muscles, weakness, and sometimes, but not often, contracture. In cancer of the spine this compression of the nerve-roots is often brought about in a peculiar way; the laminæ of the vertebra, softened by the cancer-growth, give way under the weight above them, and fall in upon the nerves lying in the intervertebral foramina. The symptoms due to direct compression of the cord are those with which we are familiar in transverse lesions: paralysis, anæsthesia, or other modification of sensation, increased reflexes, often some vesical trouble, and generally spastic rigidity of the paralysed muscles. The relation of anæsthesia to paralysis varies much in different cases, and in the same case at different times. Loss of motion is, as a rule, the most prominent symptom, and anæsthesia may be entirely absent. The activity of the reflexes is often in excess of the motor paralysis. It is another important feature, when the compression results from caries, that recovery may take place completely, or improvement may again be followed by relapse. The site of the compression, of course, determines some difference in the symptoms. Compression *limited to one side* will cause the pains to be unilateral, and the paralysis may be on the same side, the anæsthesia on the opposite, as has been stated to be the result of strictly one-sided lesions (*see* p. 262). *Cervical* compression may be accompanied by alterations of the pupil,

especially dilatation from irritation of the cilio-spinal centre, by cough and dyspnœa, dysphagia, vomiting, or very slow pulse. The distribution of the paralysis is also sometimes striking; all four limbs may be paralysed, the upper limbs being wasted, with diminished reflexes, as a result of compression of the lower motor

FIG. 36



Relations of the conus terminalis, cauda equina, and vertebræ. The vertical lines represent the course of the lumbar and sacral nerve-roots within the vertebral canal. (*After Raymond.*)

neurons. But the arms may be paralysed as a result of compression of pyramidal fibres, and the muscles will then preserve their volume and their electrical reactions, while the reflexes are increased. In some such cases the legs remain unaffected.

The distinctive features of compression of the *lumbar* enlargement of the cord, which reaches from the tenth dorsal vertebra to the first lumbar vertebra, are paralysis of the lower extremities, with flaccidity, wasting, anæsthesia, diminution of the reflexes, paralysis of the sphincters, and the formation of bed-sores.

In the lowest section of the spinal canal, which extends from the second lumbar vertebra to the coccyx, are contained the end of the spinal cord, or *conus terminalis,* and the sheaf of lumbar and sacral nerve-roots, or *cauda equina.* The greater part is occupied by the cauda equina, as the conus medullaris ends at the lower border of the second lumbar vertebra (*see* Fig. 36). The conus may be the

subject of inflammatory lesions extending from the lumbar swelling, but the majority of the lesions affecting the cauda equina result from fractures and luxations of the bones, hæmorrhages from direct injury, and pressure from tumours growing from the membranes or vertebræ.

A lesion of the conus alone causes paralysis of the bladder and of the rectum, failure of the sexual reflex, and of the Achilles tendon reflex, and anæsthesia corresponding to the third, fourth, and fifth sacral nerves (*see* Figs. 26 and 27), while power in the thighs and legs is fully maintained.

If the nerves which form the cauda equina are involved, the results will depend on the situation of the lesion. If it is at the second lumbar vertebra the whole cauda is affected. There is atrophic paralysis of all the muscles of the lower extremity, anæsthesia to the level of the groins, paralysis of the bladder and rectum, and impotence. A little less high, the ilio-hypogastric and ilio-inguinal nerves are spared, and the testicle retains its sensitiveness. A lesion below the third lumbar vertebra spares the crural and obturator nerves, and causes paralysis of the glutei, flexors of the thigh, muscles of the foot, with corresponding anæsthesia, and vesical, rectal, and sexual paralysis. A lesion of the sacral canal below the exit of the second sacral nerves injures the lowest three sacral nerves and the coccygeal nerve, and the results are the same as those of a lesion of the conus. Lower lesions may spare the sexual apparatus, and the bladder and rectum, and the lowest will involve only the coccygeal nerve.

Some observations make it likely that the sexual centres are higher in the conus than those of the bladder and rectum, and that the centre for erection is higher than that for ejaculatio seminis.

Lesions of the cauda equina are more likely to be accompanied by severe pains, those of the conus by anæsthesia or paræsthesia.

The **Diagnosis** of compression-paraplegia depends largely on the pain indicative of nerve-compression in association with the weakness resulting from pressure on the cord. Often the spine is tender at the seat of disease, and the back should always be examined for the prominence which caries so often produces (*kyphosis*, angular curvature). It has been already stated that the compression is not generally dependent upon the curvature, but on the inflammatory products about the diseased bone; and, as a fact, all the symptoms of compression may precede by several months the appearance of any projection of the spine.

Prognosis.—Even without operation, improvement may occur in cases of caries, and recovery takes place sometimes after months or years.

Treatment.—This consists in the removal of the cause. The treatment of tumour has been mentioned; cancerous growths in the spine are beyond surgical assistance. Paralysis from caries requires prolonged rest, with sea air, nutritious diet, cod-liver

oil, and tonics; and when improvement is well established, the spine should be supported by a plaster-of-Paris or felt jacket for some time afterwards. In some cases, however, the pus and caseous inflammatory products have been successfully removed by the operation of *laminectomy*.

DISEASES OF THE MEDULLA OBLONGATA

The medulla oblongata, or bulb, is subject to similar diseases with other parts of the central nervous system, such as hæmorrhage, inflammation (bulbar myelitis), and tumours. The symptoms are determined by the anatomical structure of the medulla oblongata, which, besides transmitting the motor and sensory tracts, contains the special nerve-centres of the lower cranial nerves, from the fifth to the twelfth. Hence, on the one hand, there may be paralysis of the trunk and limbs; on the other, impairment of the functions of phonation, articulation, mastication, and deglutition. These symptoms make up what is commonly called "bulbar paralysis"; and it will be best to describe first a chronic form known as progressive bulbar paralysis, and subsequently the more acute results of hæmorrhage and embolism, and the effects of the growth of tumours.

PROGRESSIVE BULBAR PARALYSIS

(*Labio-glosso-laryngeal Paralysis*)

In this disease there is a slowly developed paralysis of the lips, tongue, larynx, and pharynx, resulting from degeneration of the nuclei of the nerves which supply the muscles of these parts.

Ætiology.—It occurs in middle and advanced life, between the ages of thirty and seventy, and is more frequent in men than in women. It is not always possible to attribute it to any cause; but falls and other injuries involving the neck, and syphilis have occasionally been credited with its production. Bulbar paralysis is intimately related to progressive muscular atrophy and amyotrophic lateral sclerosis, occurring often as the last stage of either of those diseases; and sometimes an illness which has begun as bulbar paralysis has at a later date affected the spinal centres.

Symptoms.—The disease is generally first evident in the movements of the *tongue*, and the articulation of sounds which depends upon it becomes faulty. These are, first of all *e*, and then *s*, *l*, *k*,

g, *t*, *d*, *n*, *r*, and *sh*. The paralysis increases, and may become so complete that the tongue cannot be protruded, but lies always at the bottom of the mouth. After a time atrophy takes place, and the organ becomes wrinkled and furrowed. Fibrillary contractions are often observed in it. Shortly after the tongue begins to be paralysed, the same change occurs in the *lips*. The articulation of *o*, *u*, *p*, *f*, *b*, *m*, and *v* is impaired, and whistling, blowing, and pouting are performed with difficulty. The lower lip drops away from the teeth, the naso-labial folds are more marked, and saliva dribbles from the angles of the mouth. Atrophy here also can be detected in the lips becoming thinner, and fibrillary contractions may be seen. Food also collects between the teeth and the cheek; but the paralysis of the facial nerve is confined to the lower half of the face, the upper half remaining entirely free. After the tongue and lips, the *palate* is paralysed, and as a result, liquids may regurgitate through the nose, and the voice acquires a nasal quality. The articulation of *o* and *p* is also impaired by this, since the volume of air which is required for their production is diminished by its escape into the nasal cavity. Paralysis of the *larynx* produces hoarseness, and, finally, complete aphonia; and during swallowing, food is apt to enter the larynx from the paralysis of the tongue and certain muscles of the larynx (arytænoideus, thyro-ary-epiglottideus, and thyro-arytænoideus externus), by the combined action of which the two passages should be cut off from one another. As a result, choking takes place, and small particles are inhaled, which may set up bronchitis or lobular pneumonia. The laryngeal paralysis also renders coughing and hawking difficult or impossible. Dysphagia is further aggravated by paralysis of the *pharyngeal* muscles.

The progress of the disease is very slow, but the condition of the patient in an advanced stage is highly characteristic. The lower lip falls; from the angles of the mouth dribbles saliva, which the patient is constantly wiping away with a pocket-handkerchief; the tongue cannot be protruded, and the only sound uttered by the patient is a hoarse grunt as the air is forcibly driven through the flaccid glottis. With all this, the intelligence and memory, appetite and digestion, the functions of the bladder and rectum, are perfect, and, as a rule, the special senses, the movements of the eyes, the sensibility of the skin of the face and mucous membrane of the mouth, and motor and sensory power in the limbs are unaffected. Only in rare cases does the disease extend to higher centres, so as to produce deafness or ocular paralysis, or to affect the sensation of the face; and if motion of the limbs is affected, it is by the lesion of progressive muscular atrophy or amyotrophic lateral sclerosis. The electrical excitability of the affected muscles is, in part at least, retained, but in advanced cases is much diminished. Erb says that the muscles of the chin, lips, and even the tongue, show a marked reaction of degeneration, while the electric irritability of the nerves is normal or but slightly diminished. The reflexes are generally

diminished, so that the palate, pharynx, or larynx may be irritated without exciting retching, vomiting, or coughing; but they sometimes persist till late in the disease, and Erb describes reflex contractions in the muscles of the chin and lips. Fever is absent, vaso-motor disturbances are not necessarily present, nor has glycosuria or albuminuria been observed as part of the disease. Occasionally, towards the end, the pulse becomes very rapid (140 to 160). Death takes place by exhaustion from inanition, by choking, by dyspnœa or sudden cardiac failure, or by bronchitis, pneumonia, or gangrene set up by the inhalation of particles of food.

Pathology.—To the naked eye the medulla oblongata may show but little, or there may be some want of symmetry, or slight shrinking; or, on section, discoloration or blurring of the outlines. Changes are more obvious in the nerve-roots proceeding from the medulla; those of the hypoglossal and facial, the vagus and accessorius, are gray in colour and shrunken, and the microscope shows that a number of fibres are atrophied and degenerated. Microscopic examination of the corresponding nuclei reveals degeneration and atrophy, or complete disappearance of the nerve-cells, some increase of the neuroglia, and thickening of the vessel-walls. These changes are most marked in the hypoglossal nucleus and the lower part of the facial nucleus, and then in the vago-accessorius nucleus. Less commonly the glosso-pharyngeal nucleus may be affected, and rarely the nucleus of the sixth nerve, and that of the motor division of the fifth.

The atrophied muscles present appearances identical with those seen in progressive muscular atrophy.

Diagnosis.—This is generally quite easy, from the chronic course and the limitation to the bulbar nerve specified. Tumours growing in or compressing the medulla are mostly accompanied by other symptoms, such as headache, noises in the ears, deafness, sickness, or convulsions. Bilateral lesions situated more centrally (*i.e.*, in the motor tracts nearer the cortex of the brain) may cause paralysis of the same nerves, but the symptoms on the two sides may not run parallel, and there will be no atrophy, electrical changes, or loss of reflexes, showing that the nerve-nuclei are intact; further, the limbs will probably be paralysed.

Prognosis.—This is absolutely unfavourable, and the duration is rarely more than three years.

Treatment.—Drugs are of no value in controlling this disease. The important thing is to secure proper nutrition, and to prevent the inhalation of particles into the lungs. It may become necessary to feed the patient by an india-rubber tube, which the patient can himself pass down the pharynx into the stomach, and liquid food can then be poured into a funnel connected with the free end of the tube. Sometimes, in the early stage, solid food in large boluses can be more easily swallowed than liquid, being less liable to pass into the larynx or nares. Galvanism has been recommended, applied both to the seat of the disease and to the wasting muscles. The

influence in the former situation—reached by placing one pole on each mastoid process—must be very doubtful; but the muscles of the lips and tongue may be galvanised, and the act of swallowing may be assisted by galvanising with the anode on the nape of the neck and the kathode on the side of the larynx. Atropine has been given to lessen salivation. The general health of the patient must be, as far as possible, maintained.

ACUTE BULBAR PARALYSIS

Contrasting with the chronic progressive form, there occur, occasionally, cases in which the symptoms of bulbar paralysis come on suddenly, or at least rapidly, as a result of *hæmorrhage, embolism,* or *acute inflammation* of the medulla oblongata.

Ætiology.—Bulbar hæmorrhage is more frequent than spinal hæmorrhage, less so than cerebral hæmorrhage. It occurs in the same circumstances as the latter. The same may be said of embolism or thrombosis of the arteries of the medulla oblongata; here, however, the distribution of the vessels becomes of interest, since, according to Duret, the nuclei of the hypoglossal and accessory nerves are supplied by the anterior spinal and vertebral arteries, those of the vagus, glosso-pharyngeal, and auditory nerves by branches of the upper end of the vertebral arteries; and the nuclei of the facial, trigeminal, and three oculo-motor nerves by branches of the basilar. These anatomical associations may serve sometimes to distinguish vascular obstruction from hæmorrhage, otherwise not always easy to discriminate. Cases of an inflammatory nature are closely related to acute encephalitis (*see* p. 348) and to acute anterior poliomyelitis (*see* p. 274), and may have the same infective causes.

From progressive bulbar paralysis this form differs not only in the rapidity of onset, but in the greater frequency of premonitory symptoms, in the irregularity of the symptoms, and in the accompanying paralysis of the limbs; since the lesions are not necessarily restricted to motor nerve-nuclei as in the "progressive" cases, but are more or less indiscriminate, affecting the motor and sensory tracts as well.

Thus with paralysis of the tongue, difficulty of articulation, and inability to swallow, there may be paralysis of all four limbs. The occurrence of the lesion on one side will produce a more or less unilateral distribution of the symptoms. On the other hand, crossed paralysis may take place; for instance, paralysis of the arm on one side, and of the leg on the opposite, from a lesion in one half of the medulla affecting the lowest pyramidal fibres going to the opposite limb just before their decussation and the highest fibres coming to the same side just after their decussation. Or a hæmorrhage situated higher in the medulla may damage the facial nerve-fibres or nucleus on the same side, and the pyramidal tract of the same side, before

its decussation to the opposite, producing a crossed hemiplegia such as results from lesions of the pons Varolii (*see* p. 324). Sometimes also there is severe respiratory disturbance, or rapid and irregular pulse, or vaso-motor derangement, shown by rise of temperature. Albumin and sugar have been noticed in the urine. There may, however, be little time for the observation of such symptoms, as, especially in hæmorrhage, the patient may fall down suddenly, with or without cry, and death may take place at once. In other cases there are headache, vomiting, noises in the ears, and epileptiform convulsions. In fatal cases the temperature sometimes rises to 107° F. or higher.

Acute bulbar myelitis is usually less rapid in its occurrence than the other lesions; vertigo, headaches, pain in the muscles of the back, may precede the more obvious bulbar symptoms. The limbs may be paralysed from implication of the pyramidal tracts. The temperature is sometimes raised, and the pulse is mostly rapid. Death takes place in from four days to two or three weeks.

Treatment.—This must be similar to that of the same lesions in the brain and spinal cord.

COMPRESSION AND TUMOURS OF THE MEDULLA OBLONGATA

The medulla oblongata may be slowly compressed as a result of caries of the occipital bone or of alterations of its shape, by enlargement of the odontoid process, by tumours such as gumma of the dura mater, growths on the choroid plexus, aneurysms on the vertebral or basilar arteries, and lastly, perhaps most frequently, by tumours of the cerebellum.

The rare occurrence of disease and rupture of the transverse ligament is followed by sudden and fatal compression of the medulla by the odontoid process. Tumours in the medulla oblongata are comparatively rare; they include tubercular masses, glioma, glio-sarcoma, myxoma, and fibroma.

Symptoms.—In compression, the symptoms characteristic of bulbar lesions may be preceded by those of irritation, such as pain in the distribution of the fifth nerve, and twitchings in muscles supplied by the facial. Convulsions, vomiting, hiccough, and dizziness may also be present; and, later, the special lesions of the bulbar nerves, and probably weakness in the limbs. The symptoms may begin on one side and spread to the other. Tumours in the substance of the medulla are not accompanied by irritative symptoms; but headache, vomiting, and convulsions may occur. Probably optic neuritis is only observed in the case of tumours sufficiently large to implicate the pons Varolii.

DISEASES OF THE BRAIN

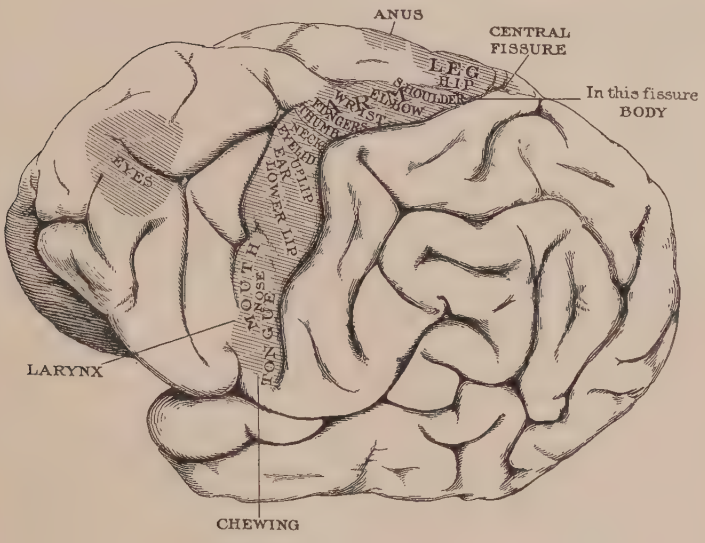
What has been said of the nervous system in general is especially true of the brain—namely, that the symptoms of disease are largely determined by the locality of the lesions, and to a much less extent by their nature. A knowledge, therefore, of the localisation of the functions of the brain is eminently desirable in the study of its diseases, and some remarks on this subject, with an account of the result of lesions, of whatever kind they may be, upon the different parts of the brain, must precede the description of the diseases which produce them. At the same time it must be recognised that, though the symptoms of a purely destructive lesion correspond accurately to the area destroyed, a tumour will, as it grows, press first upon adjacent parts, and ultimately upon every part of the encephalon, and hence the symptoms may be in excess of those which correspond to the primary seat of the lesion. Thus cerebellar tumours often press upon the motor tracts in the bulb; and occipital tumours upon the cerebellum. An irritative lesion also will produce effects which may on some occasions correspond to the actual site of the lesion, and on others will be such as to show that the irritation has spread to adjacent parts. Thus convulsions started by a lesion in the arm centre will sometimes spread to the leg of the same side, or even to the limbs of the opposite side.

LOCALISATION OF FUNCTIONS AND EFFECTS OF LESIONS

Motor Centres and Tract.—The greatest importance and interest attach to the position of the motor centres. It has been shown that electrical irritation of a certain area of the gray matter on the surface of the brain produces movements of different parts of the body, according to the spot irritated; and these experimental results are confirmed by pathological evidence, certain (irritative) lesions causing convulsions, and other (destructive) lesions causing paralysis, in the corresponding parts. In accordance with this is the fact that the Betz cells, the largest pyramidal nerve-cells (upper motor neurons) are found exclusively in the gray matter of this area. The *motor area* (*see* Figs. 37 and 38) has been held to include the convolutions about the fissure of Rolando (central sulcus), the ascending frontal (pre-central), the ascending parietal (post-central), the superior parietal lobule, and the posterior part of the marginal convolution, which corresponds to the upper ends of the central convolutions (para-central lobule). But in researches upon the chimpanzee's brain Sherrington and Grünbaum found that the motor area did not

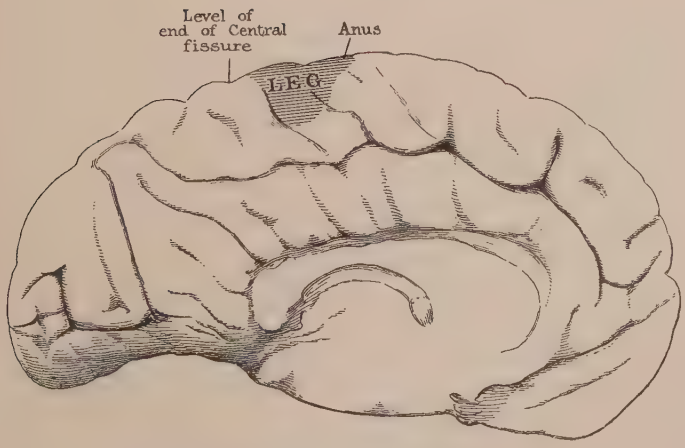
Diagrams illustrating Position of Motor Centres in Chimpanzee.
(*After Sherrington and Grünbaum.*)

FIG. 37



Outer Surface of Left Hemisphere.

FIG. 38



Inner Surface of Left Hemisphere.

extend behind the fissure of Rolando, that is, the ascending parietal or post-central convolution was not excitable. In this motor area, the centres for movements of the *face* lie lowest, or farthest from the middle line, in the lower and middle portion of the pre-central and posterior extremity of the third frontal. The centres for the *arm* lie next, and nearer to the middle line, in the upper part of the pre-central convolution, and the posterior end of the superior frontal; the centres for the *leg* occupy the upper end of the pre-central convolution close to the middle line, and a part of the marginal convolution on the inner surface of the hemisphere; while centres for the movements of the trunk are found between those of the arm and the leg. The above authors give the following as the sequence of special areas from below upwards: tongue, mouth, nose, lips, ear, eyelids, neck, hand, wrist, elbow, shoulder, chest, abdomen, hip, knee, ankle, toes, perineal muscles, anus and vagina. These areas extended irregularly forward into the frontal convolutions, and were not limited by any particular sulci.

Fibres from the motor convolutions gradually converge through the white matter of the centrum ovale to the base of the brain, and there pass between the lenticular nucleus and the optic thalamus, occupying the genu and the anterior two-thirds of the posterior limb of the internal capsule. Thence the pyramidal fibres pass into the lower part of the crus cerebri, at first somewhat on the outer side, and then in the middle; and they can be traced through the pons Varolii into the medulla oblongata. Here a *decussation* takes place, the greater part of each pyramidal tract crossing to the opposite lateral column, and a small portion remaining on the same side at the inner margin of the anterior column, forming the column of Türck, the fibres of which successively decussate lower down in the cord. The pyramidal fibres, the axons of the upper motor neurons, ultimately terminate in the anterior cornua, by arborisations about the cell-bodies of the lower neurons (*see* p. 196).

A sufficiently large destructive lesion of any part of the pyramidal fibres between the motor centres and the pons Varolii causes *hemiplegia*, or paralysis of the face, arm, and leg, on the *opposite* side of the body. This is a common result of a hæmorrhage into the internal capsule; but disease above this, where the fibres are spread over a wider area, may lead to a more limited paralysis. Extensive lesions of the cortex, such as suppurative meningitis, or meningeal hæmorrhage, will also cause complete hemiplegia, but smaller lesions may occupy only particular centres, and give rise to the form of paralysis called *monoplegia*. Thus a disease of the facial centres results in paralysis on the opposite side of the facial muscles alone, *facial monoplegia;* and in a corresponding manner there may be a *brachial monoplegia*, or a *crural monoplegia*. If the adjacent centres of the face and arm are involved together, *brachio-facial monoplegia* is the result, and if those of the arm and leg together, a *brachio-crural monoplegia*. But the centres for the face and leg can scarcely be affected at the same time, without those for the arm, which lie

between them, being involved; and, as a fact, a *cruro-facial monoplegia* has never been recorded.

Sensory Tract.—The relations of the sensory neurons in the brain have been mentioned (*see* p. 197). Much of the sensory tract must pass through the posterior third of the posterior limb of the internal capsule, since this part has been found to be injured in cases of anæsthesia of the opposite half of the body (*hemianæsthesia*). The sensory centres are now generally believed to be located in the post-central convolution and in the adjacent parts of the parietal lobules, the areas for the face, arm, and leg holding the same relative positions as the motor centres for these parts. Lesions may cause loss of sensation, more in the distal than in the proximal parts: irritative lesions cause numbness and tingling, but attacks beginning with those signs generally involve motor functions also.

Frontal Lobe.—The posterior extremities of the upper two frontal convolutions, adjacent to the pre-central, contain, according to Ferrier, centres for rotation of the head and eyes; the posterior extremity of the third frontal convolution is connected on the left side with speech movements, so that a lesion there situated causes motor aphasia (*see* p. 333). The remaining portion of the frontal lobe seems to be related to the intellectual functions, and lesions may lead to loss of memory, of the control of emotions, and of the powers of attention, of association, of ideas, and of judgment.

Parietal Lobe.—In the angular gyrus and supra-marginal lobule are visual centres; and lesions of these produce the condition known as word-blindness, in which words are seen but not understood (*see* p. 333). A lesion behind the ascending parietal convolution, somewhat higher than the supra-marginal, will cause astereognosis (*see* p. 203).

Temporal Lobe.—The upper convolution contains the centre for hearing, and destructive lesions produce deafness on the side opposite to the lesion. A lesion on the left side in a right-handed person may cause word-deafness, in which words are heard as sounds, but their meaning is not understood (*see* p. 335). Irritative lesions of the superior gyrus cause subjective sensations of sound, those of the lower end of the temporal lobe, subjective sensations of smell and taste.

Occipital Lobe.—This contains cortical centres for vision, and their destruction on one side causes hemianopia of the opposite, with unimpaired central vision and normal pupillary reaction (*see* p. 224). Removal of both lobes causes total blindness.

Corpus Striatum.—Lesions of this nucleus cause either no symptoms, or only such as are due to implication of the adjacent internal capsule. Its relation to pyrexia has been mentioned (*see* p. 28).

Optic Thalamus.—The posterior part of this gray mass contains the central ends of some optic nerve-fibres, and lesions may produce complete hemianopia. Disease of the optic thalamus has also been present in cases where hemiplegia has been followed by athetosis or

choreic movements. Experimental lesions have affected the sensory functions, especially the sense of the position of the limbs and common sensibility, but not pain, or the sense of temperature.

Internal Capsule.—This is the layer of white matter lying, in front, between the lenticular nucleus and the anterior end of the caudate nucleus; behind, between the lenticular nucleus and the optic thalamus. These two portions are not in one vertical plane, but join at an obtuse angle, opening outwards. The anterior portion is stated by Brissaud to contain fibres connected with the intellectual functions; at the angle (or *genu*) pass fibres connected with the movements of the eye, the head, the tongue, and the mouth; and through the posterior limb, run the remaining pyramidal fibres (anterior two-thirds), and the sensory fibres (posterior third). The pyramidal fibres have the following arrangement from before backwards: next to the fibres for the mouth are those for movements of the shoulder, and then successively those for the elbow, hand, abdomen, hip, knee, and foot. The internal capsule is frequently involved in cerebral hæmorrhage, and paralysis of the limbs of the opposite side is usually the result of it. Hemianæsthesia may be present if the posterior fibres are affected.

Pituitary Body.—The nervous symptoms which may result from a tumour of this organ are explained by its pressure upon adjacent parts. The most characteristic are double temporal hemianopia from pressure on the inner side of each optic tract, the hemiopic pupil reaction (*see* p. 224), and some ocular paralysis. Acromegaly is sometimes associated with these symptoms.

Corpora Quadrigemina.—A lesion of these bodies causes reeling ataxy and double ophthalmoplegia; but experiments on monkeys appear to show that such symptoms can only be due to injury of adjacent structures, since destructive lesions limited to the ganglionic masses of the quadrigeminal bodies were not productive of any obvious permanent phenomena (Ferrier and Turner).

Crus Cerebri.—Paralysis of the limbs on the opposite side and of the third cranial (oculomotor) nerve on the same side is characteristic of a lesion in this part. Lesions of the *mid-brain* (which includes the corpora quadrigemina and the crus cerebri, with the substantia nigra and red nucleus) have not infrequently been accompanied by definite, rather slow tremors of the limbs (G. Holmes).

Pons Varolii.—This portion of the brain contains the pyramidal tracts, and the nuclei of the fifth, sixth, and seventh nerves. Large central lesions may paralyse all four limbs from the proximity of the two tracts to the middle line. A one-sided lesion in the upper part produces hemiplegia of the ordinary type (*see* p. 326), on the opposite side of the body; but a lesion in the lower part, while involving the same pyramidal fibres for the arm and leg, is below the facial fibres for the opposite side, and destroys the facial nerve-roots of its own side. There is then produced a variety of hemiplegia known as *crossed hemiplegia*, the face being paralysed on the side of the lesion, but the arm and leg on the opposite side.

Cerebellum.—Lesions of the hemispheres are not always accompanied by symptoms unless they are of such a nature and size as to press upon the subjacent pyramids of the medulla oblongata, or such as to involve the median lobe. In the latter case occurs a characteristic form of ataxy (*see* p. 370), vertigo, nystagmus, especially on lateral conjugate deviation of the eyes, and sometimes convulsions of tetanic and opisthotonic character. This receives an explanation from the facts that the cortex of the superior vermiform process contains the central ends of the cerebellar tracts, of Gowers' tracts, and of fibres from the nuclei gracilis and cuneatus; that fibres crossing the middle line connect the roof nuclei of the cerebellum with the nucleus of Deiters, which is closely associated with the labyrinth of the ear; and that efferent fibres proceed from Deiters' nucleus to the third and sixth nerve nuclei, to the antero-lateral tracts, and to the anterior cornua of the spinal cord (A. Bruce).

Doubtful Regions.—There are some parts of the brain, known also as silent areas, or latent regions, the destruction or irritation of which *may* produce no distinctive phenomena. Of cortical regions, the frontal lobe, part of the inferior parietal, most of the right temporal lobe, and some of the left temporal lobe are of this nature: and in the deeper parts, the corpus striatum, the optic thalamus and portions of the centrum ovale.

THE ARTERIES OF THE BRAIN

In connection with the frequency with which hæmorrhage, embolism, and arterial thrombosis are causes of cerebral disease, the distribution of the arteries of the brain has much importance.

The *anterior cerebral* artery supplies the superior frontal convolution, and the inner surface of the hemisphere nearly as far back as the occipito-parietal fissure. The *middle cerebral* artery supplies the greater part of the outer convex surface of the brain, all, indeed, except the superior frontal and the occipital convolutions; that is, it supplies the second and third frontal, the parietal, and upper and middle temporo-sphenoidal gyri. The *posterior cerebral* supplies on the outer surface the occipital lobe, and all the inner or tentorial surface from the limit of the anterior cerebral distribution to the tip of the temporal lobe, which is supplied by the middle cerebral. The *vertebral* and *basilar* arteries supply the cerebellum and the pons Varolii.

The central parts of the brain, including the great ganglia, are supplied by branches, which come off from the circle of Willis and the origins of the three cerebral arteries, pass vertically into the nerve substances, and do not anastomose with each other or with the arteries of the cortical system. These are divided into groups: one, the *internal striate*, supplies the two inner parts of the lenticular nucleus, and the anterior part of the internal capsule; another, the *lenticulo-striate*, supplies the outer part of the lenticular nucleus; and

a third, the *lenticulo-optic*, supplies the anterior part of the thalamus. One large vessel in the lenticulo-striate group was called by Charcot the *artery of cerebral hæmorrhage*, from the frequency with which its rupture was the cause of apoplexy.

The relations of the central ganglia to the larger arteries are as follows: The *caudate nucleus* derives its supply from the anterior and middle cerebral arteries; the *lenticular nucleus* from the anterior choroid, anterior cerebral, and middle cerebral; the *optic thalamus* from the posterior communicating central branches, and from the posterior cerebral; the *corpora quadrigemina* and *geniculate bodies* from the posterior cerebral; the *internal capsule*, in its anterior half from the anterior cerebral and middle cerebral, in its posterior half from the posterior communicating, anterior choroid, and middle cerebral arteries.

HEMIPLEGIA

Before dealing with the diseases of the brain it is desirable to describe in more detail the form of paralysis known as hemiplegia.

Though this term seems to imply paralysis of one half of the body, as a fact some muscles are not paralysed in ordinary cases of hemiplegia, and of those that are paralysed some suffer much more than others. Paralysis is most marked in the arm, leg, and face, on the side opposite to that of the lesion; the muscles of the trunk, chest, and abdomen are but little affected, and the ocular muscles not at all. It is stated that, on careful testing, some weakness of the muscles on the apparently unparalysed side can also be shown.

Even in the face, arm, and leg, many differences exist. In severe cases the arm and leg may be completely motionless, but in milder cases the leg is less paralysed, and in course of recovery the leg nearly always gets better first. The facial muscles are never paralysed to the same extent as they are in a lesion of the nucleus or trunk of the facial nerve (*see* p. 235); the upper half of the face is always less affected. Thus, the eye can always be shut, and the forehead wrinkled, though it may be obvious that the frontalis and the orbicularis are not so strong as those on the opposite side. In the lower part of the face the paralysis is marked: if the patient is told to smile, or to show the teeth, the angle of the mouth is drawn up on the healthy side, and on the paralysed side it remains unmoved or is drawn inwards. But these paralysed muscles under the influence of emotion will contract more powerfully than they do when stimulated by voluntary effort. Sometimes there is a slight degree of paralysis of the tongue shown by the tip being turned, during protrusion, to the paralysed side by the unbalanced action of the healthy genio-hyoid and genio-hyoglossus muscles. The action of the respiratory muscles varies with the nature of the movement. In many cases during ordinary breathing, the upper part of the chest on the paralysed side moves more than that on the other side;

H. Jackson thinks this is a result of injury to fibres between the cortex and medulla on the opposite side, which act as continuous inhibitors of the respiratory centre. But during forced inspiration the movement on the paralysed side is less than that on the other. The weakness of the abdominal muscles on the paralysed side may be shown when the patient coughs; and by the divergence of the umbilicus to the unparalysed side when he tries to sit up. But paralysis of the spinal muscles is not commonly observed, and the muscles moving the eyeball, the muscles of mastication, and those of the larynx are unaffected. The explanation commonly adopted for these variations in the paralysis is as follows. It is first to be observed that the parts that are least paralysed, or not paralysed at all, are those which rarely or never act independently of their fellows on the opposite side; whereas the parts that are most paralysed are much more independent, and may be capable of performing acts, of which the corresponding muscles on the opposite side are incapable. As an extreme instance may be mentioned the eyes, of which one never moves except in association with the other; their muscles are not affected. In contrast with these are the hands, of which the right may be able to do things the left cannot, and *vice versâ;* these parts are most affected. There is of course a close association by commissural fibres both in the brain and in the spinal cord between the two sides: and we must suppose that, when particular movements involve synchronous (or alternating) actions of the same muscles on the two sides of the body, such as the ocular movements, phonation, and locomotion, the commissural connections have become functionally active in the highest degree, so that in the case of a lesion of the brain on one side, the corresponding muscles can be stimulated by the opposite undiseased centres; whereas in unilateral movements the commissures are not functionally active, and no help can be derived from the healthy centres. In other words, the bilaterally associated muscles are represented in and can be stimulated by both hemispheres, whereas the muscles acting independently are excited by the opposite hemisphere only.

Causes of Hemiplegia.—This form of paralysis may arise from any lesion involving, either directly or by pressure, the pyramidal tract from the cortex to the upper part of the pons Varolii. If the lesion is mainly cortical, it must be sufficiently extensive to include the centres of the face, arm, and leg. With these on the left side, the motor speech centre may be involved, when *aphasia* will result; and if a right hemiplegia occurs without aphasia it is possible that the cortical lesion is nearer the middle line, and then the leg may be more paralysed than the arm. The most frequent causes are hæmorrhage, embolism, and thrombosis, involving cerebral vessels; others are infective encephalitis, and more localised abscess; tumours; meningitis, whether suppurative or tubercular; meningeal hæmorrhage; injury to the surface of the brain, as from blows, or from compression of the infant's skull during birth, whether by

forceps or not (*birth-palsies*); and the condition known as porencephalus.

Course and Associated Conditions.—The duration of hemiplegia is very variable. It may get completely well in the course of a few weeks, the power of movement being gradually restored in the face first, and the leg afterwards, so that the patient can walk about, while the arm is still useless. Sometimes, however, recovery progresses up to a certain point during the first two or three months, until a stage is reached beyond which no improvement takes place. The paralysis is then generally accompanied by rigidity or contracture of the muscles, which gradually develops during the first few weeks. The fingers become flexed into the palm of the hand, and the elbow is slightly bent; any attempt to overcome the flexion is resisted, and causes considerable pain. In the lower extremity the knee is only slightly flexed, and the foot is often extended at the ankle. With this so-called *late* rigidity—to contrast it with the rigidity of the apoplectic state described below (*see* p. 339)—the deep reflexes are increased, the knee-jerk is greater, and ankle-clonus is readily obtained. The muscles may lose a little of their bulk from disuse, but are not degenerated, and the electrical reactions, faradic and galvanic, remain normal. This condition is precisely similar to what is seen in lesions of the pyramidal fibres (upper neurons) in the spinal cord, *i.e.*, a spastic paralysis. In the same way the rigidity and increased reflexes have been attributed to the secondary degeneration of the lateral tract; but the increased reflexes, at least, are present from an early stage of the paralysis, before degeneration can have become well established. Babinski's sign (*see* p. 204) is present in such cases. More rarely there occur in the partially paralysed muscles other disorders of movement, of which the most important are *athetosis* and *post-hemiplegic chorea.*

In *athetosis* there is a constant involuntary slow movement of the fingers, which are abducted, adducted, flexed, and extended in the most irregular way. Similar, but generally less extensive, movements may affect the arms and the toes (*see* Fig. 39). In *post-hemiplegic chorea* the movements are more violent, jerking, and ataxic, and affect the arm and leg as much as the fingers and toes. Both contractures and post-hemiplegic movements are very intractable.

The occurrence of these post-hemiplegic disorders of movement is especially frequent when hemiplegia arises in early life, either at birth or within the first ten years (*infantile hemiplegia*), and then often as a result of encephalitis. The paralysis frequently recovers up to a certain point, when some muscles become rigid and others become the subjects of athetosis or post-hemiplegic chorea. This is most marked in the arm, which is flexed at the elbow, with the hand dropped, and the fingers and thumb twisting and writhing. The leg of the same side is stiff, with slight flexion of the knee, talipes equino-varus, the tendo Achillis rigid, and the toes moving about in a purposeless manner. The gait is limping, and the pelvis is tilted to give room for the swing of the stiff leg. Another

important feature is this, that as the child grows the affected limbs do not keep pace with the others, and the arm may be found years after to be one and a half or two inches shorter than its fellow, while the hand is narrower and altogether more delicate in form. The legs show similar but less marked differences. The muscles of

FIG. 39

One of the Positions which the Hand may assume in Athetosis. (*After Turner and Stewart.*)

the affected arm and leg may be less bulky than those of the opposite side, but are never decidedly wasted; and they show no diminution of the electrical reactions or reflexes, thus differing from the muscles in acute poliomyelitis. Sometimes the muscles are extraordinarily hypertrophied, probably from the constant involuntary contractions. In some cases convulsions occur in the paralysed limbs; in others the patients are dull, stupid, epileptic, or idiotic; but in others, again, the mental condition is perfectly normal. Some of these cases are described as *infantile spastic hemiplegia.*

A pronounced *anæsthesia* is not common with hemiplegia, and if it occurs with the onset of the attack it generally passes off within a few days. It is on the same side of the body as the motor paralysis. Dr. Gordon, of Philadelphia, says that in nearly all cases there is some affection of cutaneous sensation, but its complete loss is uncommon, and hyperæsthesia in any form is still rarer. The sense of pain is diminished most of all (*analgesia*), that of temperature is less so, and the sense of touch still less. *Astereognosis* (*see* p. 203) is also often present. These changes are more marked in the upper limb than elsewhere. Occasionally there is a complete *hemianæsthesia,* affecting equally the face, arm, leg, and trunk up to the middle line of the body, as well as the special senses, so that

the patient is unable to smell, taste, hear, or see on that side of the body. The affection of sight is a real blindness of one eye, or amblyopia, and not loss of one half of the visual field, or hemianopia (*see* p. 224).

Conjugate deviation, or forcible rotation of the head and eyes to one side, is occasionally associated with the sudden or apoplectic onset of hemiplegia: it may continue after consciousness has returned; but it generally subsides after a few days or a week. Ferrier has found that there is a region of the cortex situate in the *frontal* lobe, irritation of which causes deviation of the head and eyes; but pathological results show that conjugate deviation is not restricted to lesions of one locality alone. There is, however, an important connection between the position and the nature of the lesion and the side to which the deviation occurs. When the lesion of the *left* side of the brain occurs, giving rise to *right* hemiplegia, the eyes and head are turned to the *left* side; that is, the eyes are turned *away from the paralysed side,* or the patient is said to *look towards his lesion.* But if, as a result of this lesion, there should occur convulsions in the paralysed limbs, or if a cerebral lesion of any kind causes convulsions on the opposite side with rotation of the head, neck, and eyes, the rotation will be *towards* the convulsed side—that is, the patient will appear to *look away from his lesion.* And, similarly, with right-sided lesions, there will be left-sided paralysis with right-sided deviation, or left-sided convulsion with left-sided deviation. Always, then, in *cerebral* lesions, deviation with paralysis is away from the paralysed side, deviation with convulsion is towards the convulsed side. So far as the nerve mechanism is concerned, the following explanation of the phenomena was given by Ross: For purposes of vision on any one side of the body, the external rectus of that side receives the first stimulus and the internal rectus of the opposite side is at the same time innervated, not directly from its cortical centre, but by commissural fibres between the nucleus of the third nerve and that of the sixth. With increased stimulus to see to the right, the rotator muscles of the head and neck (deep muscles on the right side, and sterno-mastoid on the *left* side) are brought into action, being also innervated by commissural fibres between the sixth nerve-nucleus and their own. Thus a cortical or supra-nuclear lesion on one side stimulating the centre of the opposite sixth nerve will cause convulsive deviation of the opposite eye to the opposite side, away from the lesion; and the other associated muscles will follow suit. On the other hand, a destructive cortical or supra-nuclear lesion on one side will paralyse the opposite sixth nerve, and with it the nerve to the internal rectus of the same side as the lesion, and the nerves to the muscles which rotate to the opposite side; and hence the rotation of these parts by the action of antagonistic muscles *towards* the lesion. The explanation seems to me much less satisfactory as regards the paralytic deviation than as regards the deviation with convulsion. Spasm of its antagonist is not the necessary,

nor even the usual, immediate result of paralysis of any muscle; and in the paralytic cases, the deviation of the head is forcible and spasmodic, and it resists all ordinary efforts to replace it in a median position.

Lesions in the *pons Varolii* may also cause deviation, but the results are the converse of the above; because at this level a lesion which damages the pyramidal fibres corresponding to the opposite arm and leg will involve the sixth nerve (and nucleus) to the external rectus of the damaged side. With a destroying lesion the deviation is towards the paralysed side and away from the lesion; with a convulsing lesion the deviation is away from the convulsed side and towards the lesion.

Mental symptoms are not very infrequent accompaniments of hemiplegia from whatever cause. There is often confusion of mind, loss of memory, difficulty in fixing the attention, and emotional weakness, so that the patient readily cries or laughs, especially the former. If speech is affected (*aphasia* or *anarthria*) the mental condition may be difficult to appreciate: it will probably appear defective, and according to some, the occurrence of an *aphasia*, involving loss of memory for words, must of itself impair the mental processes.

This form of loss of speech requires separate consideration.

APHASIA

The term *aphasia* means loss of speech from a cerebral lesion, and must be distinguished—

(1) From *aphonia*, or voicelessness, which is due to failure of the laryngeal muscles, and is not indeed a loss of speech, since words can be uttered by whispering;

(2) From *anarthria*, or defect of articulation, which is due to imperfect action of the muscles of the lips and tongue, consequent upon lesions of the medulla oblongata or the nerves proceeding thence (bulbar paralysis);

(3) From purely *mental* aberrations independent of demonstrable lesion of the cerebral centres.

With rare exceptions, aphasia is due to a lesion on the left side of the brain, and consequently, if it is associated with hemiplegia, it is with a *right hemiplegia* and not with a left hemiplegia. The only explanation of this is that the left side of the brain is alone or chiefly educated for speech purposes; and this view receives support from the rare cases of aphasia associated with left hemiplegia, in which it has often been found that the persons were left-handed. It is thus suggested that the side of the brain which is educated for the most extensive use of its associated opposite arm also develops the functional activity of its speech-centres, while those of the opposite side are comparatively inactive, though there is good reason to believe that they have some share in speech-processes. For instance,

cases of *crossed aphasia* have occurred in which a left-handed person has had aphasia in association with a right hemiplegia.

In aphasia, then, the muscles of the lips are used perfectly so far as the utterance of any letter or even of any syllable is concerned. The words which the patient can speak are clear, distinct, and natural, or if words and syllables are mixed unintelligibly, it is obviously from imperfection of the higher centres or from want of perfect automatism in them, rather than in the action of the muscles of articulation. There is none of the blurred utterance or thick speech of bulbar paralysis, general paralysis, or alcoholism. But the possible defects are very numerous, and are as follows:

The patient may be able to utter no word at all (*aphemia*); or he can only say a few words, such as "yes" or "no," or give his name; or he may have the use of some half-dozen words, which he gives in answer to every question. He may be unable to give names to any object shown him. In some cases, if the patient is told the name, he appears not to recognise it, and certainly cannot repeat it; in other cases he recognises it at once and repeats it. Some patients are utterly unconscious of the unfitness of the words they use for the ideas they seem to want to express; others perceive at once their mistakes, and manifest, as a rule, considerable annoyance thereat. In some cases the patient talks freely, but some words are skipped, others are repeated, and the whole is incoherent: this is called *paraphasia*. If aphasia coexists with right hemiplegia it is not surprising that the patient should be unable to write; but even if there is no paralysis, or it has quickly recovered, the patient may be unable to write intelligibly or at all, conditions which are called *paragraphia* and *agraphia*. In particular cases also there may be found defects in such allied functions as the use of signs or gestures, the appreciation of musical sounds, and the exercise of intelligence in general.

The various defects may be conveniently arranged for investigation of any case in the following order, assuming, of course, that the patient is neither blind nor deaf. He may be unable (*a*) to hear words spoken; (*b*) to understand words spoken; (*c*) to see words written and printed; (*d*) to understand words written and printed; (*e*) to speak from memory; (*f*) to repeat words; (*g*) to read aloud, *i.e.*, to speak from sight; (*h*) to write from memory; (*i*) to write from dictation, *i.e.*, from words heard; (*k*) to write from a copy, *i.e.*, from words seen; (*l*) to name familiar objects seen, heard, or felt; (*m*) to recall to mind objects named; (*n*) to remember the uses of things seen.

Speech depends not only upon perfect co-ordinating and motor (outgoing) processes, but also very largely upon the functions by which the materials for speech are supplied, and these are the senses of hearing and of sight.

And it may be here pointed out that the child learns to speak by attempting to imitate words which it first hears, and that sub-

sequently the process is very much aided by printed and written words and by objects which are seen.

The following are generally accepted as the parts of the brain concerned in the faculty of speech: (1) An auditory speech centre, in the upper extremity of the first left temporo-sphenoidal convolution, in which audible words are perceived, and, it is believed, their images are stored; (2) a visual speech centre, in the occipital lobe and adjacent left angular gyrus, by which visible, written or printed, words are perceived, and in which their images are stored; (3) a motor speech centre, in the posterior part of the third left frontal convolution, or Broca's convolution, which stimulates the muscular apparatus to utter speech sounds; (4) a motor writing centre, probably in the posterior part of the second left frontal convolution, related to the muscles of the hand, for the purpose of writing; (5) commissural fibres between these. There is no evidence of a centre for names (Broadbent) or of an ideational (psychical) centre above all and in commissural connection especially with (1) and (3), as assumed by some.

Lesions of any of the four centres or of the commissural fibres between them will cause some form of aphasia or agraphia. If the motor speech centre is destroyed there is *motor aphasia.* If either the auditory or the visual speech centre is destroyed there is *sensory aphasia.* A lesion may be large enough to involve two or more of these centres, or commissural connections rather than centres may be injured, so that the different combinations of aphasic conditions which may result are very numerous. Every cause of hemiplegia (p. 327) may be a cause of aphasia; an obstruction of the Sylvian artery is probably the most frequent, but a lesion of the visual centre may be due to trouble in the posterior cerebral artery.

Motor Aphasia.—The patient is speechless, or has but few words at command, and these are often only words like "yes" or "no," or interjections, or words used emotionally, as oaths; he cannot utter words spontaneously, nor can he repeat. But he can understand what is said to him, his sensory centres being unaffected; and he is perfectly aware of any errors that he makes, if he says anything at all. Thus he is deficient in respect of *e, f,* and *g.* In most cases there is *agraphia* (*h, i, k*) at the same time. Sometimes the patient can write, but cannot copy. He may also have *alexia,* or inability to read so as to understand (*d*), and this has been thought to result because the motor speech processes are so much employed in the act of reading. A pure motor aphasia (*fronto-capsular,* Dana) is rare, because the area involved is supplied by more than one artery.

Sensory Aphasia.—This occurs in two forms, according as the ingoing processes concerned are those of sight or hearing.

In the former case, the patient suffers from *word-blindness* or *alexia.* He cannot see or understand words printed or written, cannot read aloud, and cannot write spontaneously from dictation or copy, as writing is stimulated directly from the visual centre; but

he may be able to talk well, and his intelligence is good. His defects are *c, d, g, h, i, k.* It is possible in different cases to have blindness to *words,* blindness to *letters,* and blindness to *numerals,*

FIG. 40

Situation of lesion in the first left temporal convolution, causing Word-deafness. (*After Charcot, Bouchard, and Brissaud.*)

either independently of the others, and probably each due to a very localised lesion within the visual speech centre. Some cases in youths of great difficulty in learning to read have been attributed

FIG. 41

Situation of lesion causing Word-blindness. (*After Charcot, Bouchard, and Brissaud.*)

to a *congenital* word-blindness. The lesion is in the visual area, in the occipital lobe, and adjacent parietal region: and hemianopia is frequently present.

In a variety of word-blindness (*sub-cortical alexia*) the lesion is below the cortex, cutting through commissural fibres only. The patient can then write voluntarily and from dictation. In these

cases also, or in any lesions of the visual speech centre deep enough to involve the optic radiations of Gratiolet, the connections of the centre with the retina are injured, and the patient has hemi-

FIG. 42

Situation of lesion in case of Motor Aphasia. F. Ascending frontal convolution. F^3. Third frontal convolution. P. Ascending parietal convolution. (*After Charcot, Bouchard, and Brissaud.*)

anopia of the side opposite to the lesion (right and lateral hemianopia).

If the auditory speech centre is primarily involved, the patient

FIG. 43

Situation of lesion in case of Agraphia. (*After Charcot, Bouchard, and Brissaud.*)

has *word-deafness*. He can hear ordinary sounds, and may recognise and distinguish musical tones and melodies, but he cannot recognise and understand words spoken to him, cannot repeat words, and cannot write from dictation. He cannot recall words, so that his intellectual and reasoning faculties are seriously affected; and

objects presented to the other senses (sight, touch) do not call up the words or names associated with them. As the auditory word-images are essential both for perfect written and spoken matter, he speaks, reads aloud, and writes imperfectly, using wrong words and syllables—*paraphasia* and *paragraphia*. Thus he is defective in *a, b, d, f, g, i, k*.

If the lesion extends into the parietal lobe (angular gyrus, and supramarginal gyrus), it constitutes a parieto-temporal aphasia (Dana), or sensory aphasia of Wernicke.

Some other conditions may be associated with sensory aphasia; they are *mind-deafness*, or inability to recognise the meaning of any kind of sound; *tone-deafness* or *auditory amusia*, in which musical sounds and melodies are not recognised or distinguished; and *mind-blindness*, or inability to recognise the meaning or use of any kind of object. The word *apraxia* has also this meaning.

Dana points out that the most common form of aphasia is a mixed form, which he calls *fronto-lenticular*, or *lenticular*. There is inability to talk, or even to say a word (aphemia), inability to read (alexia), and inability to understand complicated sentences. It is associated with decided hemiplegia. The lesion involves the branch of the middle cerebral which supplies the corpus striatum, internal capsule, and fibres converging from the second and third frontal and precentral convolutions; apparently also the association tracts from the occipital lobe.

Recovery, or at any rate improvement, in speech may result from education of the opposite hemisphere—a very slow process—or from stimulation of the motor centre by the opposite hemisphere in cases where the lesion is in the motor-path, below the speech centre itself, and where the callosal fibres connecting the speech centre with the opposite side are untouched.

Beside the organic lesions, such as embolism and hæmorrhage, which more commonly produce aphasia, functional disturbances may cause it, such as migraine and right-sided convulsions. Hysteria more often leads to aphonia from laryngeal failure.

The modern theory of aphasia is challenged by P. Marie, who regards it as a disturbance of the intellectual functions from a lesion of Wernicke's area (supra-marginal, angular, and upper ends of first and second temporo-sphenoidal gyri). Lesions of this area cause sensory aphasia; lesions in front of it involve the lenticular region, causing defective articulation or anarthria; lesions of both areas cause motor aphasia, or aphasia of Broca. A pure alexia results from lesion of the lingual and fusiform lobes; aphasia is added if the lesion extends into Wernicke's area.

CEREBRAL HÆMORRHAGE

In dealing with diseases of the brain prominence must be given to diseases of the vessels of the brain, which are responsible for the majority of cases of cerebral paralysis. Rupture of the vessels, with escape of blood (*hæmorrhage*) into the brain, and obstruction of the arteries by *embolism* and by *thrombosis*, are the forms of vascular lesion which have to be considered.

Ætiology.—Cerebral hæmorrhage occurs more frequently in men than in women, and more in advanced life than in youth. Thus it occurs, but is very infrequent, below the age of thirty, and has, indeed, occurred in one as young as nine; but nearly four-fifths of the cases occur after the age of forty. A large proportion have granular kidneys and hypertrophied hearts; the arteries are generally at the same time atheromatous, sclerosed, or calcareous, late results of endarteritis; and frequently the actual cause of hæmorrhage is the rupture of minute (miliary) aneurysms, which are found on the branches of the cerebral arteries. Alcohol, gout, and syphilis have their share in the production of these arterial lesions. Hæmorrhage is sometimes associated with heart disease and endocarditis, and this is especially the case when it occurs in quite young people. Probably in these instances an embolism has been the first lesion, around which the artery has softened, and then it has ruptured; or the artery has yielded so as to form an aneurysm which has subsequently burst—a process similar to what will be described as occurring in the hæmorrhage from the lung in phthisis. Hæmorrhages often occur in the substance of tumours of the brain, and they may be so large as to render their source uncertain: smaller hæmorrhages take place in conditions of general tendency to bleeding (scurvy, purpura), and as a local result after ligature of the common carotid artery.

Seats of Hæmorrhage.—Scarcely any part of the brain is exempt from the risk of hæmorrhage, but it is much more frequent at the base in the neighbourhood of the corpus striatum and optic thalamus, which are mainly supplied by the branches of the middle cerebral artery, to which reference has already been made (*see* p. 325). Vessels may also burst in the lateral ventricles (*ventricular hæmorrhage*), or on the surface of the brain (*meningeal hæmorrhage*); but when blood is found in these situations, it has often proceeded from a hæmorrhage primarily in the cerebral substance.

Anatomical Changes.—In different circumstances the blood effused may be small in quantity, or amount to several ounces. In the latter case it tears up the brain tissue, destroying, for instance, the great ganglia, and the internal capsule, and extending thence into the centrum ovale; or it may burst through the optic thalamus or caudate nucleus into the lateral ventricle. Thence the blood flows by the aqueduct of Sylvius into the fourth ventricle. Such cases are rapidly fatal, and *post-mortem* examination reveals a mass

of black clot, filling the ventricle and occupying much of the hemisphere, surrounded by brain tissue, which is ragged and discoloured by blood. The pressure exerted by the clot is shown by one, or even both, hemispheres being enlarged, with flattened convolutions and closed sulci. In cases which have lasted a few days there is the same black-red clot, and the brain tissue around is soft and discoloured yellow, from absorption of hæmoglobin (*yellow softening*). In later stages the clot becomes brown, or brownish-yellow, consisting of disintegrated blood and nerve-tissue; and the surrounding tissue is frequently softened (*white softening*), and contains granule corpuscles. Finally, in patients who survive, the blood becomes absorbed, and leaves a tawny or orange-coloured spot, in which crystals of hæmatoidin can be found; or a cyst may remain, containing serous fluid; or a distinct, tough, fibrous scar, discoloured also by the remains of blood pigment.

Secondary Degeneration.—Permanent lesions of the pyramidal tract, or of the cortical motor area, are followed by descending secondary degenerations, like those which occur in disease of the spinal cord. Such degenerations follow the course of the pyramidal fibres below the lesion; thus, a lesion of the internal capsule causes this change to take place in the middle third of the crus cerebri, in the anterior part of the pons, in the pyramid of the medulla oblongata on the same side, in the column of Türck, also on the same side, but in the posterior part of the lateral column of the spinal cord for its whole length on the opposite side. Lesions of the corpus striatum or optic thalamus alone are not followed by secondary degeneration.

Course and Symptoms. *Apoplexy.*—Cerebral hæmorrhage may be preceded for days or weeks by occasional giddiness, numbness or twitching of the fingers, headaches, insomnia, or some diminution of mental capacity; but these are not so much indications of the severe attack to come as evidences of existing disease of vessels, and perhaps due themselves to slight hæmorrhages. On the other hand, it may come on without any warning whatever. Sometimes, also, it seems attributable to a definite cause, such as emotional excitement, muscular effort, violent coughing, or straining at stool; but at others it occurs when the patient is perfectly quiet, or even during sleep. In numerous cases cerebral hæmorrhage causes a group of symptoms known as *apoplexy*—that is, the patient is struck down suddenly unconscious, or he quickly becomes so.* As a fact, it is rarely that a patient is absolutely struck down and unconscious in a moment; but it does happen, and patients have died in five or ten minutes from the first symptom. More often the symptoms come on slowly. The patient is seized with intense pain in the head, becomes faint or slightly collapsed, may be sick or have a

* The word apoplexy (ἀποπλήσσω—to strike to earth) has been sometimes applied to the hæmorrhage itself; but hæmorrhage is only one of its causes. The terms pulmonary apoplexy and renal apoplexy, for hæmorrhages into the lung or kidney, are obviously undesirable.

slight convulsion; and then, after half an hour or so, gradually sinks into a condition of coma. This order of events has been called *ingravescent apoplexy*. Or the first symptoms may show themselves in the motor system: the patient mumbles in his speech, or his arm drops powerless, and he gradually leans over to one side, falling if not supported, and then lapses by degrees into coma. Or the coma may be developed in a few hours through stages of increasing drowsiness. Occasionally the attack begins with convulsions, or vomiting occurs as an early symptom. When the patient is found by the friends alone, or is picked up in the street unconscious, or is unable to be roused in the morning from sleep, it is of course impossible to say what the onset has been. But undoubtedly cerebral hæmorrhage may occur without apoplexy; a very slight bleeding into the motor tract alone may give rise to paralysis without loss of consciousness.

The patient suffering from hæmorrhagic coma lies completely unconscious, and cannot be roused by shouting or any form of stimulation of his skin. The face is flushed, the pulse is full and tense, the breathing is *stertorous*, a loud snoring noise being made in consequence of the palate or tongue falling back and impeding the passage of air into the chest. The condition of the limbs varies: both legs and arms may be quite flaccid, falling at once when raised; or it may be obvious that the leg and arm on one side are more flaccid than those on the other. The muscles of the face share in the paralysis, and the cheeks are puffed out and sucked in with the processes of respiration; this may also occur only on one side. Sometimes, however, the limbs of one or both sides are in a condition of rigidity (*early rigidity*), the muscles contracted and resisting extension or flexion. The deep reflexes are commonly increased, and the skin reflexes are absent. The pupils are variable; they are sometimes contracted, at others dilated or unequal. Conjugate deviation may occur. The temperature shows a slight fall, which may continue till death, or if life is prolonged it rises a little above the normal. Sugar and albumin are occasionally found in the urine, from pressure on the medulla oblongata. In very severe cases, the pulse and breathing are rapid, there is profuse sweating, and intense flushing of the face and skin generally; then, after a time, two or three hours or more, the patient becomes livid, râles occur in the larger bronchi and trachea, the pulse gets weaker, the breathing slower, and finally death takes place. The fatal termination may, however, be delayed for several days, during which the lungs are very apt to suffer from œdema or pneumonia; and the occasional passage of particles of food or fluids through the glottis probably contributes to the inflammation of these organs. In more favourable cases, the patient lies simply comatose, with but little disturbance of his pulse or respiration, and gradually regains his senses in the course of a few hours or two or three days. In a large proportion of cases, the patient is then found to be suffering from *hemiplegia*, which may itself slowly recover or be permanent (*see* p. 326).

Diagnosis.—This has to be made from embolism, from uræmia, from alcoholic intoxication, and from other conditions causing apoplexy on the one hand and hemiplegia on the other. It will be best considered after cerebral embolism has been described.

Prognosis.—This is generally unfavourable in proportion to the extent or severity of the first symptoms. That apoplexy is often fatal is well known, and death may take place at different intervals after the onset. The cases are grave in which the coma is profound, with much stertor, flushed or congested face, full bounding pulse, and complete relaxation of all the limbs.

If, after recovery from the first coma, headache continues, and the patient again becomes drowsy, the result is likely to be serious, and probably inflammatory changes are taking place about the clot, or in the area of softening.

Treatment.—In an attack of apoplexy, the patient should be in the recumbent position, with the head and shoulders slightly raised. Stertor is often diminished by placing him on one side, and this is further beneficial by allowing the full play of at least one lung, viz., that which is uppermost. An ice-bag should be applied to the head. If the bowels are known to be confined, or not recently opened, a drop of croton oil or a few grains of calomel may be placed on the tongue, or an enema of castor oil or turpentine may be administered. If the coma continues the catheter may be required to empty the bladder. Venesection is rarely carried out now, and is not generally desirable, though formerly it was the universal practice. Occasionally rapid improvement under the use of bleeding, or of leeches applied to the temple, has been seen, but the nature of such cases must be doubtful. In later stages, pain in the head requires the continued application of ice; the patient should be carefully nursed to prevent the formation of bed-sores, and a water-bed may be needed. If hemiplegia occur without loss of consciousness, the patient should be kept quiet in bed, the bowels should be attended to, and a light diet of fish, milk, custard pudding, &c., should be enforced for some days or weeks. Neither here, nor in the cases beginning with coma, can the treatment of the paralysis be entered upon until all evidences of active mischief in the brain have subsided. Generally, after six weeks or two months the paralysed limbs may be gently faradised; but if headache, dizziness, or other unpleasant sensations are caused by this treatment, it must be delayed yet longer. As the muscles contract to faradism, this form of electricity may be employed, with the object of maintaining the muscles in the highest state of efficiency possible to them under the circumstances. If contractures show themselves, passive movements, frictions, and faradisation of the antagonists of the contracting muscles should be employed. The possibility of a second attack should be remembered, and the patient should have always a light simple diet, should abstain from alcohol, take only moderate exercise, and keep from business or other mental worry as much as possible.

EMBOLISM AND THROMBOSIS OF CEREBRAL ARTERIES

Pathology.—The usual cause of *embolism* is mitral or aortic endocarditis; the former is far more common. In either case particles of fibrin are detached from the surface of the valves, or in the case of a contracted mitral orifice, fibrin may be deposited in the dilated left auricle, and subsequently detached and carried into the cerebral vessels. The middle cerebral artery is more often obstructed than others, and the left oftener than the right; the reason for these differences is not clear.

Thrombosis is most frequently caused by disease of the vessel wall, such as atheroma, by which the surface is roughened, and fibrin is consequently deposited. Syphilitic disease of the arteries produces considerable narrowing of their channels, and thus favours thrombosis. In addition, thrombosis may occur from several conditions weakening the circulation, such as those resulting from enteric fever, typhus, cancer, phthisis, and other severe illnesses.

Embolism and thrombosis, by obstructing the circulation of the blood, alike lead to *softening* of the districts of the brain to which the vessels correspond, unless the vascular supply is maintained by means of anastomoses. These are not abundant in the case of the cerebral vessels, and, indeed, the vessels going to the central ganglia are really terminal vessels, while those going to the cortex of the brain anastomose more or less. At least, this is true of the distribution of the middle cerebral artery—the vessel most often obstructed. A part of the brain in which softening has taken place has generally lost the smooth, glistening surface of a normal brain-section, is more opaque, or gray, or speckled; it breaks down readily under a stream of water; or it is milky, or diffluent. It shows under the microscope drops of myelin, portions of nerve-fibres, granule-corpuscles, and free fat-globules. It sometimes has a yellowish or brownish colour from altered blood-pigment; or minute extravasations of blood may be present in cases of sudden obstruction, and a form of *red softening* results. In cases of rapid death after embolism, the brain substance may look perfectly healthy, as there has not been time for any changes visible to the naked eye to take place. Occasionally an embolus sets up inflammatory changes in its neighbourhood; sometimes it leads to aneurysm and cerebral hæmorrhage as already described (*see* p. 337). Rarely actual infarcts are formed. The later stages of softening consist in the absorption of the disintegrated tissue, and the formation of a cyst; or, if the softening is small, a cicatrix may be produced.

Embolic lesions, involving the motor tract, are followed by the same secondary changes (descending sclerosis) as are hæmorrhagic lesions. A persistent lesion of the brain, whether embolic or hæmorrhagic, causing hemiplegia in infancy or early childhood, has the remarkable effect of checking the growth of one half of the

brain, or it may be of other parts of the central organs, so that years after it is smaller than the other half, and is described as atrophied (*cerebral hemiatrophy, unilateral atrophy*). If the lesion is in the motor cortex, the hemisphere is atrophied on that side, and there is sclerosis of the pyramidal tract: if it is in the basal ganglia, there is in addition atrophy of the middle fillet in the pons and medulla, and of the antero-lateral region of the spinal cord on the *same* side; and atrophy of the cerebellum, superior cerebellar peduncle, and dentate nucleus on the *opposite* side (Mott and Tredgold). The growth of the paralysed limbs is also impaired (*see* p. 328).

Symptoms.—The results of *embolism* are not very different from those of hæmorrhage; but it more often causes sudden hemiplegia without loss of consciousness than does hæmorrhage. Obstruction of a large vessel will cause sudden loss of consciousness, and death may take place soon after. In other cases, coma comes on more gradually, and may be preceded by pain in the head. When the patient comes out of his coma, he is often found to be paralysed on one side, and if the paralysis is on the right side, aphasia may be present. Since the softening occurs only in the areas supplied by the vessel beyond the seat of obstruction, the symptoms are more likely to correspond with the distribution of the artery than in hæmorrhage, where the extravasated blood ploughs up the brain with little discrimination. If the middle cerebral be obstructed near its origin, there will be hemiplegia of the opposite side; and if the lesion is on the left side, aphasia also, since this artery supplies the internal capsule, Broca's convolution, the greater part of the motor area of the cortex, the first and second temporal convolutions, and the angular gyrus. Persistent hemiplegia is accompanied by the conditions already mentioned (*see* p. 328).

Thrombosis is usually less rapid in its effects, though with the same results—apoplexy and hemiplegia; but sometimes a sudden coma occurs, indistinguishable from that of hæmorrhage. There are often premonitory symptoms—headache, dizziness, loss of memory, drowsiness, numbness, or formication of an arm or leg, or of one side of the body. Senile forms of disease are frequently of this nature. The symptoms are aggravated from time to time by fresh lesions, not necessarily confined to the motor tracts, and they are often followed by mental weakness—or dementia—the "softening of the brain" of old people.

Diagnosis of Cerebral Vascular Lesions.—This may be divided into two heads—the diagnosis of apoplexy from other conditions simulating it, and the diagnosis from one another of the different causes of apoplexy or hemiplegia.

(1) In the former the history is of great importance. Cases of coma occurring in the course of severe illnesses may be readily excluded; it is coma coming suddenly or rapidly which may be confounded with apoplexy. Fagge pointed out that *pyæmia* might occasionally be accompanied by a sudden coma closely resembling it. More commonly the conditions to be discriminated are coma

from injury, poisoning by opium, alcoholic poisoning, uræmia, diabetes, and epilepsy.

Cases of *injury*, in the absence of history, may present the greatest difficulties, as even with the external evidence of injury, it may remain uncertain whether the patient has fallen as a result of apoplexy, or has injured his brain in consequence of the fall. Even after death the problem may be insoluble. The position of a scalp wound in relation to the weaker side, if paralysis can be recognised, may sometimes help; and the age of the patient, or other circumstances of his bodily health, may render a spontaneous lesion of the brain more, or less, likely.

Opium-poisoning is generally distinguished by the minutely contracted pupils, the slow pulse, and slow respiration; but it may be closely simulated by hæmorrhage into the pons Varolii. Evidences of the unilateral lesion, such as greater flaccidity or rigidity of limbs on one side, or unequal pupils, are in favour of hæmorrhage.

The same may be said of *alcoholic poisoning*. The condition is one of profound coma, without any one-sided symptoms. Evidence of alcohol may, of course, be obtained from the breath, or from the stomach by means of emetics or the stomach-tube. But a patient may have drunk freely or sufficiently just before an apoplectic attack, or if the attack has come on gradually he may have taken a glass of spirits as treatment.

Uræmia is probably always accompanied by albuminuria; but the detection of albumin in the urine does not exclude cerebral hæmorrhage; for, in the first place, hæmorrhage occurs often in those who have granular kidneys; and, secondly, hæmorrhage may itself produce albuminuria in those who have healthy kidneys. In uræmia sometimes the coma is less profound; the patients are more easily roused for a time by shouting, to relapse again into coma. There is no paralytic weakness, no vaso-motor disturbance, and no flush of congestion, such as occurs in some cases of apoplexy. Addison used to call attention to the hissing nature of the stertor. General convulsions alternating with a coma, or a previous sudden amaurosis, are in favour of uræmia.

Diabetic coma develops very slowly, and is not profound till near the end: it is often preceded by severe abdominal pain. The pulse is rapid and feeble, the breathing is often slow, deep, or sighing, and the breath has a sweet odour. The urine is saccharine; but here also a mistake is possible, for a hæmorrhage involving the fourth ventricle may produce glycosuria.

Epilepsy is sometimes followed by coma, which is more like natural sleep than that of apoplexy; the patients are more easily roused, and there are no unilateral symptoms. Early age would be opposed to apoplexy. On the other hand, a gradual onset excludes epilepsy. Occasionally, *hysterical* patients will lie unconscious for long periods, but the cases are generally distinguished by other characteristic symptoms.

It should be noted that other organic diseases of the brain,

such as *umour* and *meningitis,* may rapidly terminate in coma, but the history will generally be sufficient to prevent mistake.

(2) In the diagnosis of the causes of apoplexy, one has to consider the nature of the attack and the associated condition of the patient. It will have been seen that the nature of the attack often gives but little help. Hæmorrhage, embolism, and thrombosis may all produce a sudden or rapid coma. The more severe and prolonged the coma the greater the probability of hæmorrhage, whereas a pronounced hemiplegia, occurring without coma or with very transient unconsciousness, is more likely to be due to embolism. Age is in favour of hæmorrhage, and youth almost excludes it unless an antecedent embolism is possible; but in persons between forty-five and sixty years of age positive indications in one or other direction are often wanting. In ventricular hæmorrhage evidence of blood will be found in the liquid withdrawn by lumbar puncture.

The associated conditions of hæmorrhage are albuminuria and other evidences of renal disease, or arterial degeneration, with tense and rigid or thickened arteries, hypertrophied heart, and arcus senilis. Senile changes in the arteries may also be recognised in many cases of thrombosis. Hemiplegia in young subjects free from heart disease is often due to syphilitic arteritis, to which, of course, the history may give a clue. In embolism there is generally a mitral or aortic murmur, or some evidence of dilatation of the left cavities of the heart, which serves as a source of the embolus; or there may be signs of embolism in other parts of the body, such as enlargement or tenderness of the spleen, blood in the urine, the characteristic appearances in the retina, or obstruction of an artery in one of the limbs.

Treatment.—If cerebral embolism can be certainly recognised, the treatment is similar to that of cerebral hæmorrhage; but depletion must not be thought of. Absolute rest, milk diet, ice to the head if there is pain, and gentle laxatives or enemas if the bowels are confined, are the main indications.

The treatment of the resulting hemiplegia is also the same. In "infantile" cases little can be done; bromides may be given when there are fits (*see* Epilepsy), and impaired gait may be assisted mechanically. The hindrance to walking from rigid extension at the ankle may justify amputation of the foot.

CEREBRAL MENINGEAL HÆMORRHAGE

Hæmorrhage in connection with the cerebral membranes may be between the bones of the skull and the dura mater (*extra-dural*) or on the surface of the brain within the dura mater (*intra-dural*). The causes of hæmorrhage in these situations are: (1) direct injury by fall or blow on the skull; (2) compression during delivery in new-born infants; (3) endarteritis and degeneration of cerebral

vessels; (4) antecedent inflammation of the dura mater (*see* Cerebral Pachymeningitis, p. 362).

Extra-dural hæmorrhage proceeds mostly from the middle meningeal artery, and is caused by direct injury producing fracture of the skull. The effused blood presses upon the brain, and causes symptoms which are dependent upon the extent and seat of pressure, such as coma and paralysis. These cases are dealt with in works on surgery.

Intra-dural hæmorrhage may also result from direct injury. The hæmorrhage in new-born infants is more often intra-dural, and forms a clot which spreads over the surface of the brain. The blood probably proceeds from meningeal veins entering the longitudinal sinus, which are torn during compression of the cranial bones. In such cases the new-born infant is drowsy and cyanosed, sucks badly or unwillingly, breathes irregularly, or has unilateral or general convulsions. A lumbar puncture shows that there is blood in the cerebro-spinal fluid. Death takes place in a few days, or, if the child survives, it may subsequently suffer from paralysis and rigidity (*diplegia spastica*), and the brain undergoes atrophy and sclerotic changes (*see* p. 342).

Hæmorrhage from disease of the vessels occurs mostly in persons of middle or advanced years, under pathological or ætiological conditions precisely similar to those of intra-cerebral hæmorrhage, but with much less frequency (*see* p. 337). The symptoms are variable, and not distinctive; and this is probably explained by the frequency with which the blood spreads itself over a large area, instead of being limited by surrounding brain tissue to a small spot. Coma is the most marked feature in these cases, and it may come on suddenly or gradually. It is sometimes preceded by such indications as headache, giddiness, or vomiting. Convulsions occur occasionally, and may be local, unilateral, or general. Paralysis and rigidity are not necessarily marked. Sometimes there is mental disturbance—either excitement, delirium, or dulness. Blood may extend on to the surface of the brain from the interior: the symptoms due to the internal lesion will generally predominate.

Lumbar puncture may assist in the diagnosis and even in the treatment of a meningeal hæmorrhage. Where a positive diagnosis can be made, an operation for removal of the blood should be considered: in infants this has been successfully done within a few days of birth (H. Cushing).

HÆMORRHAGE INTO THE PONS VAROLII

If hæmorrhage takes place into the central region of the pons, there is generally profound coma, with minutely-contracted pupils and complete paralysis of all four limbs: a condition which may easily be mistaken for poisoning by opium. Death is often very rapid, but it may be delayed some hours or three or four days.

Convulsions and vomiting are frequent, and sometimes the temperature rises to a great height before death. If a slight hæmorrhage permits of recovery, there will probably be some degree of paralysis of the limbs, with anæsthesia, irregular facial paralysis, paralysis of the tongue and articulation, and dysphagia.

Hæmorrhage into the lateral regions causes the forms of paralysis previously described (*see* p. 324), and conjugate deviation, if it occurs towards the paralysed side (*see* p. 330). If the posterior or upper surface of the pons is involved, sugar or albumin may appear in the urine, and the urine may be abnormally abundant (*polyuria*). The symptoms are, in part, the same as those which result from hæmorrhage into the medulla oblongata, since these two portions of the nervous system are continuous with one another.

CEREBELLAR HÆMORRHAGE

This is of rare occurrence, and the symptoms present little that is distinctive of the part affected. In some cases there is a close resemblance to cerebral hæmorrhage, the patient having coma with complete resolution of all the limbs, or with paralysis of one or other side. The hemiplegia is opposite to the side of the cerebellum involved, and is regarded as being due to pressure on the pons. Death may occur in a few hours, or days, or the patient may recover from the apoplexy and remain hemiplegic.

Sometimes the symptoms are more obscure, as in the case of a boy aged fifteen, which came under my notice. There was first severe pain in the head; the next day he was sick and constantly throwing himself about in bed; the following day he became lethargic, and he died in the evening. Abercrombie records the case of an old woman who was seized with sudden coma and vomiting, and died after forty hours. A clot was found in the right lobe of the cerebellum.

ENCEPHALITIS

Whereas in the case of the spinal cord the tendency of older writers was to regard every softening as inflammatory (*see* p. 263), in the case of the brain every softening was held to be degenerative (pressure or embolism), and suppuration or abscess was the only inflammation recognised. But it is now certain that there are also different forms of non-suppurative inflammation, due probably in all cases to infective organisms or their toxins, affecting often limited areas of the encephalon, and having corresponding and more or less distinctive groups of symptoms.

ACUTE ENCEPHALITIS

Ætiology.—The most important feature in the ætiology of encephalitis appears to be the influence of intoxications and infections. Among the former, chronic alcoholic intoxication holds the first place, and is responsible for many of the cases described as acute hæmorrhagic encephalitis, while in some of the other forms influenza has been often observed as an antecedent; less often and with less obvious connection other infectious diseases, such as scarlet fever, measles, influenza, pneumonia, diphtheria, syphilis, gonorrhœa, and erysipelas. Injury is a cause of encephalitis, probably by facilitating infection.

Pathology.—The inflammatory change may occur in all parts of the brain, but in some forms it is almost limited to the gray matter (*polio-encephalitis*), whereas in others it has a wider distribution; the gray matter may be that of the cortex or of the basal ganglia, or that which surrounds the third and fourth ventricles; whence it may extend to the gray matter of the cord.

As a result of inflammation the colour of the gray matter becomes gray-red, violet, or dark brown-red, and the white matter becomes reddish, or pink, or gray-red; it is often finely speckled with red points of hæmorrhage. Moreover, the brain is swollen, prominent above the section, infiltrated with serum, moist and shiny. Microscopically there are hæmorrhages, near the vessels, and round-cell infiltration. The nerve-cells are pale with a swollen nucleus and turbid contents, and later they may atrophy or undergo fatty or calcareous changes; the nerve-fibres show swelling and varicosities of the axon. Influenza bacilli, pneumococci, and other organisms have been found in the inflamed area in different cases.

Associated with the hæmorrhagic form of encephalitis there is often pachymeningitis hæmorrhagica, and the pia mater is injected with blood.

Symptoms.—These may be described under the following forms:

Polio-encephalitis acuta hæmorrhagica superior (Wernicke).—This is a lesion of the gray matter about the third ventricle and of that extending back from this point to the fourth ventricle as low as the sixth nerve nucleus. The onset of the symptoms is generally sudden, and they consist of somnolence, or it may be unrest, excitement, or delirium, headache, giddiness, vomiting, and stiffness of the neck. There is ocular paralysis, and double ophthalmoplegia is a marked feature, though the sphincter pupillæ and levator palpebræ superior are spared; and there is optic neuritis or hæmorrhage into the disc. The gait is staggering, reeling, or uncertain, like the ataxy of drinking; the speech is trembling and hesitating; the pulse rapid and the temperature normal or subnormal. Death may take place in from ten to fourteen days.

This form of polio-encephalitis may be accompanied by multiple neuritis.

Polio-encephalitis acuta inferior.—Here the lesion affects the lower part of the medulla oblongata, and the symptoms are mainly those of a bulbar paralysis such as have been already described (*see* p. 318) —namely, paralysis of the face, tongue, and palate, with dysarthria and dysphagia, but with more or less extension upwards or downwards in different cases. Such cases are much more often due to infectious disease than to chronic alcoholism, and have been not unfrequently observed in the course of influenza epidemics, though the anatomical proof of their nature has often been wanting in cases that recover.

The symptoms of this condition may be combined with those of Wernicke's form, thus constituting a *polio-encephalitis superior et inferior*. In other cases, again, the lesions about the third or fourth ventricle have been combined with an extension to the spinal cord —*polio-encephalo-myelitis*. In these the lesions have not always been symmetrical, nor always confined to the central gray matter, and sensation has been sometimes involved; the cases are generally acute or sub-acute.

In some cases ataxia of cerebellar type is the chief symptom; and in a patient of my own, aged four years, who had recently had whooping-cough, there was ataxia of the arms and legs, with tremor of the trunk and head, nystagmus, and indistinct speech. Polio-encephalitis superior and polio-encephalitis inferior are, no doubt, closely related to polio-myelitis anterior acuta in ætiology and pathology, the immediate cause being thrombosis of minute vessels, followed by peri-vascular exudation, minute hæmorrhages, and small-celled infiltration. The symptoms are then determined by the position of the lesion, the first including mental changes (frontal area), hemiplegia (motor centres) or ataxia (cerebellum); the second involving the cranial nerve-centres; and the third producing the familiar symptoms of atrophic spinal paralysis (*see* pp. 273, 274).

Acute primary hæmorrhagic encephalitis (Strümpell) is another form, which is especially liable to occur after infectious diseases, and has even been called influenza encephalitis. It involves the cerebral hemispheres, but is not confined to the gray matter. It occurs in youth, or even in early childhood, affecting females especially. There is an acute onset, and when it follows influenza there is a distinct interval between the two events. The symptoms are headache, giddiness, nausea, sickness, sleepiness, and prostration; then suddenly a rigor, intense headache, vomiting, occasionally convulsions with rigidity of the neck or limbs, and generally some fever. Either at the onset, or more frequently after the first stage of dulness, occurs hemiplegia, or paralysis of an arm or leg, or aphasia, or conjugate deviation of the eyes and face. The duration is from three or four days to two or three weeks, and it is mostly, but not always, fatal. It is probable that in some cases of infantile hemiplegia the lesion has been of this kind.

Acute inflammation of the brain also occurs as a part of the acute disseminated encephalo-myelitis already described (*see* p. 265).

Diagnosis.—The differential diagnosis of these somewhat rare cases presents considerable difficulties, which must be met by a careful consideration of the symptoms in each. The diseases likely to be confounded with them are myasthenia gravis, tubercular and other forms of meningitis, thrombosis of the cerebral sinuses, and hysteria.

Prognosis.—This is not absolutely unfavourable: acute cases may be rapidly fatal, but recovery, partial or complete, has not unfrequently occurred. My own case recovered in three or four years, and was well at the age of thirty-three.

Treatment.—This can be little more than symptomatic. Rest in bed, cold compresses to the head, leeches to the temples or mastoid in severe cases, and purgatives, are the measures which may be employed.

CHRONIC ENCEPHALITIS

Of this as a separate disease little can be said. Wilks described some cases of *diffuse sclerosis*, or induration of the brain, which were probably inflammatory in origin (Guy's Hospital Reports, 3rd ser. vol. xxii., 1877). In one the convolutions were flattened and compressed. On section, the white matter was firm, hard, gray in colour, and encroached on the gray matter, pushing the convolutions away from one another. In another there was *meningo-cerebritis*, and the brain substance was tougher than normal. In a third case the membranes were also inflamed; the left hemisphere presented some red softening, but the right was hard, and brick-red in colour, the convolutions were swollen to twice their size, and the cortical was scarcely distinguishable from the medullary matter. Under the microscope the tissue showed an "absence of anything like nerve-structures, and appeared to consist mostly of vessels and a dimly fibrillated substance." In the case of an infant, recorded by Fagge, the brain was indurated, the white matter was of a yellowish colour, the gray matter appeared normal, but the pia mater was firmly adherent. The only histological change noted was a slight excess in the cells of the neuroglia. In other cases atrophy of nerve-fibres and increase of the neuroglia are described.

The early symptoms in Wilks' cases were stupidity, inability to speak, indifference to food, loss of sight, hearing, and memory, tingling and pain in the extremities, followed by drowsiness and unconsciousness, with ill-defined paralysis of one or other side of the body.

Chronic encephalitis, partial and probably secondary to degenerative changes, occurs in *disseminated sclerosis* and in *general paralysis of the insane.*

ABSCESS OF THE BRAIN

Ætiology and Pathology.—In by far the majority of cases abscess can be shown to be the result of direct infection by pyogenic organisms. It often arises in consequence of chronic suppurative disease of the ear. For instance, otitis occurs during convalescence from scarlatina, the membrana tympani is perforated, and there is a discharge of pus, which may continue for months or years. Ultimately, and sometimes without any apparent cause, the symptoms of cerebral abscess develop. In some such cases, the bone forming the wall of the tympanum is necrosed, the dura mater over it inflames or sloughs, and the pia mater becomes adherent; in others the bone may be healthy, and the infection seems to have been carried by channels in the bone to the interior of the skull. Even where the membranes are directly inflamed, the abscess may not be in immediate contact with them. The pus from the ear is sometimes fœtid, but not necessarily so. Another cause of abscess is disease of the nasal fossæ or frontal sinuses, and it may follow any other lesion involving the cranial bones, such as direct injury, syphilitic caries or necrosis, or tumour of the bones.

Cerebral abscesses occur in general pyæmia, and in some pyæmic cases patches of red softening have occurred side by side with developed abscesses. Another source of suppuration is occasionally seen in inflammatory lesions in the lungs, such, for instance, as phthisis, pneumonia, empyema, and especially bronchiectasis. It is possible here that particles of thrombus are carried from the lungs into the general circulation, and so to the brain. In some cases of cerebral embolism from infective endocarditis the softened tissue breaks down into a fluid indistinguishable from pus.

The position of the abscess is determined, to a certain extent, by its cause. Thus, if due to otitis, it is mostly in the temporal lobe; or in the cerebellum, if the mastoid cells are especially involved. Figures given by A. Starr show that abscess is nearly three times as frequent in the former situation as in the latter. Disease of the nose may give rise to abscess in the frontal lobes. Generally there is only a single abscess, but in pyæmia there are often two or more, situated indiscriminately. They are, however, commonly located in the white matter of the hemispheres, or of the cerebellum, and rarely in the gray matter, or at the base of the brain. They vary in size, and may reach two inches in diameter. Recent abscesses have a shreddy wall, those which are older have a definite and often thick cyst wall or capsule, composed of fibrillated, if not fibrous, tissue. The pus is mostly pale green, viscid, and acid in reaction; but in long-standing cases it becomes more mucoid still, alkaline, and of a bright green colour. Sometimes, especially when due to bone disease, it may be extremely offensive. The brain tissue outside the abscess may be softened. Though in many fatal cases the abscess is found intact, it may

rupture on the surface, and set up meningitis; or into the lateral ventricles; or it may form a communication, through diseased bone, with the tympanum, and discharge externally (*otorrhœa cerebralis*).

Symptoms.—These are often extremely obscure. The most constant is *pain*, of a continuous dull aching character; or more severe, so that the patient holds his head with his hands, or bores his head into the pillow, or cries out constantly. Exacerbations of the pain occur from time to time. The seat of pain often, but not always, corresponds to the position of the abscess. Sometimes there is elevation of the temperature, sometimes *rigors*, either occasionally or following with such regularity as to suggest malaria, and sometimes profuse perspiration; but the temperature may be very nearly normal. Convulsions and vomiting may occur. Optic neuritis is much less frequently present than in cases of tumour. Alterations in manner, dulness, listlessness, loss of memory, and emaciation are also sometimes observed. The common seat of the abscess renders localising symptoms on the side of the motor tract or nerve trunks rather improbable; but there may be ill-defined hemiplegia, or aphasia, which is said to be often of a kind in which the patient cannot give the name of an object shown to him, or cannot call to mind the object of which the name is mentioned (*optical aphasia, intercortical sensory aphasia*). An abscess in the cerebellum may cause vertigo, or some uncertainty of gait; and if cranial nerves are pressed upon, the corresponding paralysis will occur.

The duration of the symptoms is very variable; they may last for months, or they may end fatally in a few weeks. Death is often quite rapid, the patient becoming delirious, or quickly drowsy and comatose. Respiration may cease before the pulse, as in the sudden deaths from cerebral tumour.

Diagnosis.—The diagnosis of abscess of the brain is not always easy; the pain may be mistaken for neuralgia, and its rigors for malaria.

The most important factor in diagnosis is the presence of a primary cause, and chronic discharge from the ear is the most frequent of these. It must not, however, be too hastily assumed that acute head pains and pyrexia occurring in a patient the subject of chronic otitis are due to cerebral abscess, even though rigors and optic neuritis be present as well. For otitis may produce, besides abscess of the brain, subdural abscess, or meningitis, or suppuration of the mastoid cells with or without thrombosis of the adjacent veins and sinuses, or encephalitis. All of these are accompanied by severe head pains and fever; with mastoid abscess there may also be rigors. And it is important to remember, what has now been verified in numerous cases, that in mastoid suppuration there is often double optic neuritis, with an entire absence of meningitis or of abscess, as proved by *post-mortem* examination, and by recovery after simply trephining the mastoid cells. The cause is

probably thrombosis of some cerebral sinuses. This warning, however, applies almost more to meningitis than to abscess, since optic neuritis is less frequent in the latter. Suppurative meningitis—the form most likely to be confounded with abscess, since they have a common origin—is more rapid in its course, and is more likely to be accompanied by paralysis and fits: the temperature is more uniformly high, and shivering is absent; and lumbar puncture may show micro-organisms in the cerebro-spinal fluid.

Prognosis.—Many cases have been cured by evacuation of the abscess: without the help of surgery recovery cannot be expected.

Treatment.—Where an abscess can with reasonable certainty be recognised, and its locality accurately determined, the attempt to evacuate the pus should be made.

For exploratory purposes bone may be removed with the trephine or with the gouge, and a fine trocar can then be introduced. In cases arising in connection with diseases of the ear, it is desirable that before trephining, a thorough exploration and antiseptic treatment of the tympanum and mastoid cells should be undertaken, both to exclude the possibility of the symptoms being entirely due to these parts, and also to minimise the risk of meningitis during the longer operation.

Apart from surgical interference, the treatment of abscess of the brain must be purely symptomatic: the relief of pain may be attempted by local anodynes, by ice to the head, and by bromide of potassium, butyl-chloral hydrate, or even morphia, internally; large doses of quinine (5 grains every four hours) may also be given, in the hope of neutralising the septic condition.

INFANTILE CEREBRAL DIPLEGIA

(*Infantile Spastic Paraplegia, Diplegia Spastica, Congenital Spastic Paraplegia, Birth-palsy*)

This is a spastic condition of the legs, or of both legs and one arm, or of all four limbs together, occurring in infancy or early childhood, and very often actually congenital—that is, dating from birth.

The origin of the disease is cerebral, and the most common condition is one of atrophy of the convolutions, especially in the motor region, and sometimes of the cerebellum. The microscopic shows that the nerve-cells are markedly atrophied or absent, and in many cases also there is sclerosis from increase of the neuroglial tissue. The cerebral lesion is accompanied by degeneration of the crossed and direct pyramidal tracts.

A condition of atrophy of the brain, known as *porencephalus* (πόρος, a passage), is sometimes present. The name is given to defects in the cerebral convolutions in the form of cavities, which penetrate more or less deeply into the brain, and sometimes reach the ventricles. The cavities are lined with pia mater, filled with

subarachnoid fluid, and bridged over by arachnoid membrane. The condition is often congenital, and is attributed to encephalitis or to vascular disorders.

Meningeal hæmorrhage, as a result of prolonged labour or instrumental delivery, involving compression of the skull, and tearing of meningeal veins, is undoubtedly the origin of some cases of diplegia (*see* p. 345). Premature birth of itself, encephalitis, embolism, thrombosis, and hydrocephalus have also been regarded as causes.

As remoter antecedents many writers have noted maternal ill-health, including over-work, acute diseases, mental conditions, and syphilis.

Symptoms.—Nothing may be noticed at birth, but walking is very slowly acquired, and the legs are observed to be gradually more stiff; ultimately the condition is much like that seen in the spastic paraplegia of adults. The limbs are extended and rigid, there is increased knee-jerk, but ankle-clonus is not always to be obtained. Sometimes spasm of the adductors is extreme, and the legs are crossed one over another, in spite of which the child manages to walk—*crossed-leg progression*. The arms are never so rigid as the legs; there may be some stiffness at the elbow-joint, or the fingers are clenched. If they are much involved, the cases have been called *bilateral spastic hemiplegia*. More often there is a jerky movement, or a movement like chorea, or a mobile spasm like that of athetosis. I have recorded a case, syphilitic in origin, in which violent starting of the rigid limbs was provoked by a loud noise, or by a sharp tap on the head. Convergent strabismus, oscillation of the eyeballs (nystagmus), mental deficiency, or actual idiocy, and late power of walking are also present in many cases.

Treatment.—This is not encouraging. Practically the patients remain uncured, though a little improvement may be obtained by massage and manipulation, and in extreme deformities by division of tendons and by mechanical appliances.

HEREDITARY CEREBELLAR ATAXY

This disease affects members of the same family, and is hereditarily transmitted. The symptoms generally set in after puberty, and only slowly progress. The essential feature is the reeling, unsteady gait, characteristic of cerebellar disease; in addition, chorea-like movements, impaired articulation, and increased knee-jerks. Later, the limbs become spastic. The cerebellum has been found atrophied without sclerosis. It is distinguished from Friedreich's ataxy by the later age of onset, the presence of knee-jerks, and the absence of trophic disturbances and spinal deformities.

MENINGITIS

In the cerebral as in the spinal meninges, we have to distinguish an inflammation of the dura mater, or *pachymeningitis*, and inflammation of the pia mater, or *leptomeningitis*.

The pia mater appears to be much more subject than the dura mater to the influence of micro-organisms; and to these bodies nearly all forms of leptomeningitis can be traced.

The organisms most often concerned are:

(1) The *pyogenic organisms* (streptococci, staphylococci) which may invade the meninges in pyæmia, septicæmia, erysipelas, perhaps small-pox, after injury to and operations on the cranial bones, and in diseases of the ear, nose, and frontal sinuses; these cause a *suppurative* meningitis.

(2) *Tubercle bacilli*, secondary to a tubercular focus, either in the brain itself, or in some other part of the body.

(3) The *pneumococcus*, often in association with pneumonia, or as part of malignant endocarditis.

(4) The *meningococcus*, or diplococcus intracellularis of Weichselbaum, which causes cerebro-spinal fever, or epidemic cerebro-spinal meningitis, including its posterior basal variety (*see* p. 88).

(5) *Syphilis* is a frequent cause of meningitis, which is, however, generally subacute or chronic in its course.

(6) In some cases the organisms of *influenza*, *typhoid fever*, *gonorrhœa*, and the *bacillus coli communis*, have been found.

The results of bacterial invasion are seen in the effusion of lymph or of pus on the surface of the brain, often with an increase in quantity of the cerebro-spinal fluid.

There is a general resemblance to what has already been described under the head of cerebro-spinal fever, but the disease as determined by other organisms than the meningococcus presents sufficient differences to demand a full account at least of the tubercular, suppurative, and pneumococcal forms.

The usually gradual development of the symptoms in tubercular meningitis makes it desirable to describe it first.

TUBERCULAR MENINGITIS

Ætiology.—This disease occurs at all ages, but is generally regarded as more frequent in children than in adults; and it certainly affects males more than females. So far as its causation is concerned, it is constantly associated with tubercle elsewhere in the body, from which presumably infection of the meninges with tubercle-bacilli takes place. In numerous cases it arises in the course of phthisis, hip-joint disease, caries of the spine, or other tubercular complaints, and is then sometimes called *secondary*. Other (*primary*) cases, which are especially frequent in children and young people, seem to arise in persons previously quite healthy, or at

most after a few weeks' malaise; but even in these instances, after death, it is nearly always the case that some other lesion is found, such as caseating bronchial glands, or miliary tuberculosis of the lungs and other viscera, or a caseous nodule in the brain itself. There may be discharge from the ear, but, if it has any relation to the disease, it is either that it indicates general ill-health, or that it opens a passage for the entrance of tubercle-bacilli.

Morbid Anatomy.—The characteristic appearances are seen in the pia mater, and consist of the effusion of lymph and the presence of tubercles. The lymph, which is gelatinous and translucent, or opaque and gray, or grayish yellow, but rarely or never distinctly purulent, is contained in the meshes of the pia mater, especially at the base of the brain, over the optic chiasma, the diamond-shaped space behind it, and the adjacent crura and pons. From this central point it commonly extends into the Sylvian fissure on each side, along the course of the middle cerebral artery, where it may be very abundant. The surface of the hemispheres is commonly free from lymph, or is at most a little dull, or sticky, so that tubercular meningitis is often called a *basal* meningitis; but it is common to find a small patch of lymph at the top of the cerebellum, at the anterior part. With the lymph are commonly mixed tubercles, varying from mere points up to the size of millet seeds, generally gray and opaque, occasionally commencing to caseate. The tubercles are especially abundant on the branches of the Sylvian arteries and in the membranes between them. By separating the arteries with their branches from the brain, and floating them in water, the tubercles may be seen as minute thickenings upon the capillary branches. Under the microscope the smaller tubercles present aggregations of lymphoid corpuscles in the perivascular sheath; the larger tubercles may present all the characteristic features—lymphoid cells, giant-cells, and bacilli.

The relation of the inflammatory lymph to the tubercles is very variable. There may be abundant lymph in the characteristic situations, with few, if any, tubercles discoverable; there may be a good number of tubercles with very little lymph. Occasionally, cases are fatal with symptoms indistinguishable from those of tubercular meningitis, in which tubercles are found on the surface, and no evidence of meningitis. The ventricles of the brain are commonly distended with fluid (whence the old name *acute hydrocephalus*), the convolutions are flattened against the skull, the fornix and septum lucidum are generally soft, and the ependyma of the ventricle presents a granular or sanded appearance.

The cranial dura mater is not usually affected, but the spinal dura mater sometimes shows minute tubercles, and the lymph in the pia mater may extend to the cervical region of the spinal cord. The constant presence of other tubercular lesions in the body has been already noticed.

Symptoms.—These will first be described as they occur in the more common cases in children, and the differences in secondary

cases will be afterwards mentioned. There is often a prodromal stage, during which the child is out of health, restless, loses appetite, gets thin, may be occasionally sick, and has constipation. The illness begins more definitely by headache, or vomiting, or perhaps a convulsion. The headache is severe and continuous, with exacerbations from time to time; the child puts its hand to its head, and may be often crying, "Oh, my head!" or simply whining, or moaning, or occasionally uttering a sudden short shriek. With this there is a moderate degree of fever, quick pulse, excessive sensibility to light and sound, so that the child shuts the eyes, and desires to be left alone in bed; resents being disturbed, and often curls itself up in bed away from intruding friends. The vomiting does not generally last long. If the illness begins with a fit, this is not often repeated. Occasionally there is squint, and there may be diplopia quite early.

After a few days, still with severe headache, there may be slight delirium, and the patient becomes drowsy. The head is sometimes retracted, and the neck is stiff; the abdomen becomes hollowed out, or retracted, the outlines of the muscles are obvious through the skin, and the margins of the ribs and the iliac crests are prominent. For this the terms *carinated* and *boat-shaped* are sometimes used. The pulse may be slow, and is often irregular; the respirations are slow, sighing, and irregular; the temperature is still generally high, or oscillates between 101° and 103°. A tendency to vaso-motor paralysis is seen in the flushing of the face and the production of patches of redness wherever pressure is for a time applied. When the finger is drawn sharply across the skin of the forehead or abdomen, a broad red line quickly appears, and may persist five minutes or more. This condition, which is not peculiar to, but only more marked in, meningitis, is called *tache cérébrale, tache méningitique,* or cerebral streak. Even as early as this, changes may often be observed in the optic disc, which at first becomes highly vascular, and then shows definite optic neuritis. Tubercles are seen in the choroid in a small proportion of cases. Food is taken badly, and the bowels are constipated.

From this point the case may steadily go on to a fatal termination, without any fresh symptom. The drowsiness increases to coma, optic neuritis is more pronounced, the abdomen becomes more and more hollowed, the pulse more irregular, feebler, and generally quicker, the respiration may take on the character of Cheyne-Stokes breathing, and the temperature may fall more or less rapidly, or just before death go up quickly to 106° or 107°. Mucus accumulates in the bronchial tubes, and with failing pulse death takes place. But often the last two or three days are marked by local symptoms. An arm or leg, or an arm with the leg of the same side, becomes either rigid or paralysed; or there is slight facial paralysis, or squinting, or ptosis. The pupils are frequently unequal, and one or both may be insensitive to light. Frequently this stage is marked by convulsions, and these may recur several

times before death. With the development of these symptoms coma becomes more profound, and death takes place, as above shown, or the patient is asphyxiated in a convulsion.

The duration of the illness varies between ten days and three weeks from the beginning of the pronounced symptoms; but occasionally it may be four, five, or six weeks. The above course of the disease has been divided into three stages—a stage of *irritation*, one of *compression*, and the last, a *paralytic stage*. But it is not always easy to distinguish between them, and in some cases the more typical symptoms may be very little marked, coma alone being prominent.

In *secondary* tubercular meningitis the symptoms are often much more rapidly developed, and more insidious. They may, of course, be masked by those of the disease already existing. The patient may, with very little warning, become delirious, or have paralysis of a limb or of the face, or have a fit, quickly becoming comatose, and dying within a few days.

Diagnosis.—This is sometimes comparatively easy; at others difficult or impossible until quite late in the illness. The fact of meningitis is to be suspected when decided head symptoms are accompanied by fever; but with headache alone the diagnosis can rarely be conclusive. In young children, for instance, *otitis* may cause severe headache and moaning, vomiting, photophobia, and the desire to lie undisturbed. A careful examination may show that the pain is more or less localised, or that the ear or mastoid process is tender; or the use of the ear speculum may settle the diagnosis. *Enteric fever* may for some days simulate meningitis in the headache, drowsiness, and fever; but in enteric, headache rarely persists after the tenth day, and generally by that time the characteristic loose yellow stools, or the rose spots on a full abdomen, or the Widal serum-test will decide the diagnosis; which will be confirmed, as the case goes on, by the entire absence of convulsion, rigidity, or paralysis. The mistake is sometimes made in the other direction, cases of meningitis, without any prominent headache, but with flushed face, delirium, and pyrexia, being regarded as enteric fever. The most useful indications here are the irregular pulse, sighing or irregular respiration, rigidity of muscle, the presence of Kernig's sign (*see* p. 207), paralysis, convulsions, and optic neuritis. But the last occurs also in enteric fever, though rarely. Tubercle of the choroid is by no means common, and, with few exceptions, the diagnosis has to be made without it. Probably cases of *encephalitis* have been confounded with tubercular meningitis. The more sudden onset of the former and the basal distribution of the symptoms in the latter would help to distinguish them. In young children, decided cerebral symptoms accompany other acute illnesses, as, for instance, *pneumonia* and *broncho-pneumonia:* the child is often drowsy, with retracted head, and towards the end convulsions may occur. These symptoms would be quite explained by the detection of localised dulness with bronchial breathing; but

râles all over the chest would suggest the possibility of a general tuberculosis. Another condition that may simulate it to a certain extent is the *exhaustion* following upon malnutrition, bad feeding, or severe diarrhœa in quite young infants. The child is drowsy or comatose, with pale face, sunken eyes, dilated irregular pupils, and irregular, sighing respiration. It was formerly called *hydrocephaloid disease* or *spurious hydrocephalus*. It is distinguished from meningitis by the history, the absence of fever and local paralysis, the depressed fontanelle, and the speedy improvement under restorative and supporting treatment.

The distinction of tubercular from suppurative meningitis rests largely upon (1) the absence of local cause for a suppurative meningitis, such as cranial injury or otitis; (2) the previous existence of tubercular lesions such as phthisis; (3) the paralyses of cranial nerves, indicating that the meningitis is situate at the *base* rather than over the vertex; (4) the duration, which is commonly very much shorter in suppurative meningitis, even two or three days only. It is more difficult to distinguish between tubercular meningitis and the non-tubercular posterior basal meningitis of infants (*see* p. 90); that is, the localisation in some cases of tubercular meningitis may be precisely the same as is common in the other class of cases. Lumbar puncture may be the means of distinguishing the three forms. In tubercular meningitis, lymphocytes and tubercle-bacilli may be found; in suppurative meningitis, polymorphonuclear leucocytes; and in posterior basal meningitis, the meningococcus.

Prognosis.—Undoubtedly, tubercular meningitis is in nearly all cases fatal; and the prognosis may be regarded as unfavourable in proportion to the certainty of the case being tubercular, as, for instance, when the symptoms are secondary to phthisis, hip-disease, or other well-marked tubercular lesion. A certain number of patients presenting the evidences of spontaneous tubercular meningitis get well. The recovery is often slow: speech, vision, and the power of walking remain imperfect for weeks or months, thus showing conclusively that there has been a serious interference with the cerebral functions. But the tubercular nature of such cases may be incapable of proof; some, indeed, may have been encephalitis.

Treatment.—With the doubt thrown upon the curability of the disease, the subject of treatment seems reduced to small limits. Cold should be applied to the head by means of an ice-bag, the bowels should be opened, and milk must be given in small quantities frequently. Blisters to the back of the neck, iodoform ointment to the scalp, and other local irritants are of more than doubtful value. Of internal remedies, iodide of potassium is often given in doses of 3 or 5 grains to children, and the bromide in similar or larger doses may help to allay the pain in the head.

SUPPURATIVE MENINGITIS

Ætiology.—If we exclude cases of cerebro-spinal fever, a purulent meningitis is in the majority of cases the result of invasion by the pyogenic or septic organisms, and arises in circumstances similar to those which lead to abscess of the brain: that is, its common cause is a focus of disease in the immediate neighbourhood. Thus, it may follow injuries to the head, or be set up by extension of inflammation in neighbouring parts, such as otitis media, suppuration of the mastoid cells, disease of the nasal cavities, syphilitic caries or necrosis of the skull, suppurative phlebitis, or abscess of the brain. But it occurs also as a complication in some general diseases of an acute, febrile, or infective nature—pyæmia, septicæmia, malignant endocarditis, enteric fever, small-pox, and scarlet fever.

Pathology.—The inflammation chiefly affects the pia mater and arachnoid (*leptomeningitis*), the purulent effusion lying either in the arachnoid cavity (subdural), or much more frequently in the meshes of the pia mater itself. When it has spread from a diseased bone of the skull, the dura mater itself may show localised inflammation, but the extension of the disease over the brain is by means of the other membranes. Commonly, the convex surface of the brain presents a more or less extensive layer of bright yellow or green pus, which may be on both sides, or confined to one side, the side of the lesion in secondary cases. The pus frequently follows the course of the larger vessels, and dips down with the pia mater into the sulci. Though mostly affecting the upper surface of the hemispheres (meningitis of the convexity), it may extend to the base, or the pus may find its way, perhaps by gravitation, into the spinal canal. The brain tissue beneath it is commonly softened, and may present ecchymoses or minute abscesses.

Symptoms.—While there is a general resemblance to the symptoms of tubercular meningitis, the course of acute meningitis is usually much more rapid, and there is much diversity as to the prominence of particular symptoms. Where meningitis supervenes upon other acute illnesses, its features may be more or less masked. In idiopathic cases, or cases caused by chronic inflammatory lesions, like otitis, the symptoms often commence acutely with chill, or rigor, and acute pain in the head. This is generally very severe and constant, and aggravated from time to time. The patient becomes feverish, shuns light and noises, and may lie curled up in bed, resenting interference, as in the tubercular cases. Vomiting often occurs at the commencement. There may be rigidity of the muscles at the back of the neck, and the head is drawn back. The pupils are often contracted. Convulsions also may occur quite early, and may be followed by active delirium, or by drowsiness accompanied by delirium; and in later stages there is often paralysis, with repeated attacks of convulsions, generally bilateral. The paralysis

is very variable, corresponding to the situation of the effusion; from its frequent occurrence at the vertex it less often affects the cranial nerves than does tubercular meningitis, though there may be squint; but an arm or leg is often paralysed, or there may be complete hemiplegia. Sometimes there is rigidity of the paralysed limbs, or of their fellows. The pupils become dilated, and the ophthalmoscope generally reveals optic neuritis, which may develop rapidly under observation. The temperature is high, varying from 102° to 104°, the pulse is mostly rapid, respiration is sighing, irregular, or of Cheyne-Stokes type, *tache cérébrale* may be well marked, and in some cases the abdomen is retracted. The drowsiness passes into deep coma, and, finally, the evacuations are passed involuntarily, the breathing and circulation fail, mucus accumulates in the chest, and death terminates the scene. The disease is often fatal within two or three days of the first symptom, and sometimes even less; exceptionally the illness lasts longer, as in a patient under my care, who died on the twentieth day.

Diagnosis.—This presents the same difficulties as in tubercular meningitis, but the course being much more rapid, it is less often the stage of headache than the stage of coma, or delirium, that may be misunderstood. Sometimes a diagnosis has to be made when severe headache, and a quickly following coma, are the only important features of the case; or from a convulsion occurring quite unexpectedly in the course of some septic or infective disease. Where a primary source for the meningitis, such as otitis, exists, one may be easily led to a right opinion; in the absence of this, one must look for fever, or any indication of paralysis or rigidity of a limb. As compared with ordinary *apoplexy*, the hemiplegia of meningitis is often much less complete; it may, however, be a typical hemiplegia, so far as the distribution is concerned, from the meningitis involving the cortical motor area. Meningitis has to be recognised as one of the results of *chronic otitis*, and the difficulties in determining its presence in that disease have been already pointed out (*see* p. 351). The symptoms of meningitis may also be confounded with those of *abscess*, and all the more, as either may occur from disease of the ear, or of the cranial bones. The complete clinical course of fever, headache, delirium, coma, convulsions, and paralysis or rigidity, all within three or four days, is in favour of meningitis; in abscess there is more likely to be severe headache for some days before the coma—the temperature is either lower, or oscillating, with chills, rigors, or sweating. The diagnosis from *tubercular meningitis* has been already discussed. By relying too much upon the mental condition in cases of meningitis (and abscess) in young women, an unjustified suspicion of *hysteria* may for a time be entertained.

Prognosis.—The majority of cases of suppurative meningitis are fatal. What proportion, or if any at all, recover, is still much debated, because *post-mortem* evidence is not forthcoming to prove conclusively the actual occurrence of meningitis; but the prognosis

must be unfavourable in proportion to the rapidity and severity of the symptoms.

Treatment.—The application of cold to the head, by means of ice-bags, is the chief local means; with intense pain, leeches might be applied to the temple. The use of blisters, or mercurial ointment, is of doubtful value. Internally, bromide of potassium may be used to relieve pain, and mercurials and iodide of potassium may be given in the hope of influencing the morbid process. Where a syphilitic origin is certain, these should, of course, be pushed to full doses. The bowels should be kept active, and fluid nourishment should be given in small doses frequently.

PNEUMOCOCCAL MENINGITIS

The meninges may be the first part of the body to be invaded by the pneumococcus, when the disease may be called *primary;* but the meningitis is more often secondary to a pneumococcal infection of the lung, pleura, or other part. It is not uncommonly an outcome of malignant endocarditis, with which also pneumonia may be present. Out of twenty-three cases at the Children's Hospital, Great Ormond Street, seven followed empyema and purulent pericarditis, seven ear disease, and others followed pneumonia (F. E. Batten).

In symptoms and morbid anatomy it closely resembles other forms of suppurative meningitis, and, of course, in former times, some of the descriptions of the latter must have been drawn from pneumococcal cases. In these latter the exudation is generally a thick, viscid, greenish pus, which mostly occupies the vertex, and is either in the meshes of the pia arachnoid or in the subdural space. It is sometimes found at the base, and sometimes in both situations, and very generally there is some pus on the posterior surface of the cervical spinal cord.

The symptoms—namely, intense headache, vomiting, high temperature, paralysis, convulsions, and coma—are very rapidly developed, and may be fatal within twenty-four hours. Thus in a case of malignant endocarditis with pneumonia, the patient was taken with convulsions and died in twelve hours. Sometimes, however, in secondary cases the meningitis has been found, *post mortem*, without having given rise to definite local symptoms.

The diagnosis may be inferred from the clinical associations of the case, or from the rapidity of its course. It can generally be decided by a lumbar puncture, when the pneumococcus may be identified.

The **prognosis** is very bad; and **treatment** can only be conducted on the same lines as in other suppurative cases.

CEREBRAL PACHYMENINGITIS

The dura mater becomes inflamed on its surface (*pachymeningitis externa*) in consequence of injuries, or the extension of inflammation from diseased bone, otitis, or any of the causes already mentioned as leading to suppurative meningitis. The inner surface of the dura mater is also inflamed in many cases of suppurative meningitis (*pachymeningitis interna purulenta*). The symptoms in these cases, due to the inflamed dura mater, are not distinguishable from those referable to the other membranes.

A third affection of the dura mater is that known as *pachymeningitis interna hæmorrhagica*, or *hæmatoma of the dura mater*. Virchow's view that this is primarily an inflammation with subsequent bleeding into the newly formed tissue is now generally held, though it has been also thought that it might originate as a hæmorrhage with organisation into fibrous tissue, in which, again, new vessels and fresh hæmorrhages take place.

Ætiology.—Hæmatoma has been found most commonly in association with chronic insanity and chronic alcoholism; it also occurs in old people apart from these conditions, and in some local affections of the brain, such as apoplexy, softening, and tumour. A condition of general or local atrophy of the brain, with degeneration of the arteries, is common to all these conditions. The disease is much more common in elderly people, and more frequent in males than in females. A primary hæmatoma may arise from injuries to the skull, and probably from other conditions likely to produce hæmorrhage, such as chronic affections of the heart and lungs, and diseases of the blood.

Morbid Anatomy.—The inner surface of the dura mater is covered with one or more layers of membrane, soft and friable when recent, tougher and more fibrous when old; in colour brownish-red, brown, brownish-gray, yellow, or even white, and often presenting punctiform ecchymoses; while between the layers may be considerable quantities of blood-clot more or less altered by age, or collections of serum containing cholesterin crystals. The deposits are usually situate over the parietal region, near the middle line, and are bilateral in about half the cases. They may be mere membranes, or two or three millimetres in thickness, and if much blood is extravasated, the surface of the brain is depressed. J. O. W. Barratt states that an early change is the formation of filaments of fibrin in the interior of the blood-vessels, with subsequent vascular dilatation and hæmorrhage. He was unable to show the presence of micro-organisms.

The **Symptoms** are very variable. Often the condition has been found *post mortem* without any symptoms which would be explained by it; sometimes, on the other hand, a fatal apoplexy is the result of a large hæmorrhage between the membranous layers, which compresses the brain. Such an illness probably cannot be

diagnosed from other forms of cerebral hæmorrhage. But the attack may be less severe, and recovery takes place; or there are fresh seizures at different intervals. The symptoms are generally headache, giddiness, somnolence gradually increasing to coma, and twitchings or convulsions in the limbs and face of one side, followed by muscular weakness or definite paralysis. The pulse is often slow or irregular, the pupils are contracted, and there is some degree of fever. In the intervals the patient may return to his former condition, or present some impairment of the cerebral functions, such as diminished intelligence and memory, drowsiness, weakness of limbs, and headache.

Diagnosis.—Huguenin mentions as aids to diagnosis, besides the predisposing conditions, the evidence of sudden and increasing compression, the symptoms showing that the convexity, and especially the cortical motor area, is affected (unilateral convulsions, followed by paresis, absence of oculo-motor paralysis), the spread of the disease from one side to the other, and recovery after apparently severe illness. Cerebral symptoms in the interval, and one or more previous attacks, strengthen the diagnosis.

The **Treatment** should be similar to that of apoplexy.

THROMBOSIS OF THE CEREBRAL SINUSES

The blood coagulates in the cerebral sinuses either as a result of some profound cachexia, or in consequence of infection from lesions of adjacent parts.

The former causes mostly an adhesive thrombosis, the sinus being obstructed by laminated clot, without any general infection of the system. It occurs most often in infants, especially those suffering from marasmus or chronic diarrhœa. It mostly affects the longitudinal sinus. Coma, stiffness of the back, neck, or limbs, strabismus, nystagmus, and paralysis or spasms of the face, are said to occur. Distension of the veins over the forehead and temple, and epistaxis, have been described as resulting from thrombosis of the longitudinal sinus, and œdema of the mastoid process when the lateral sinus is affected; but the constancy of the symptoms is doubtful.

In adults the chlorosis and anæmia of young girls are occasional causes; but phthisis, cancer, and wasting diseases more commonly. The symptoms are similar—namely, vomiting, headache, drowsiness, convulsions, delirium and coma with nystagmus, strabismus, sometimes optic neuritis, and twitchings or weakness in the extremities. The result depends largely upon the primary ill-health, to which treatment must be directed.

Thrombosis from *local infection* is mainly caused by extension from disease of the ear, but the origin may be in the orbit, nose, mouth, pharynx, or other part from which the lateral or cavernous sinus can be reached. The *lateral* and the *petrosal sinuses* are more often

affected; and it has already been stated that the optic neuritis frequently accompanying the spreading forms of otitis is probably due to this. The symptoms resemble those of cerebral abscess, namely, fever, headache, delirium, stupor; and later local cerebral symptoms, such as paralysis or convulsions. Thrombosis of the lateral sinus may extend into the *jugular vein*, and produce a hard swelling, with more or less tenderness, or even redness, œdema, and finally suppuration, in the upper part or whole vertical extent of the neck. Thrombosis of the *cavernous sinus* causes proptosis of the eye, chemosis or swelling and vascularity of the conjunctiva, and œdema of the eyelids and root of the nose. The eyeball becomes fixed, and the optic nerve inflames.

In these infective cases, as a rule, septic particles are conveyed into the right heart, and thence into the lungs, so that a fatal pyæmia is the result.

Treatment.—In thrombosis dependent on general ill-health, this condition must be treated. If local lesions are the cause, they must be dealt with surgically. The spread of an infective thrombus down the jugular vein can be prevented by tying the vein below the clot and clearing out its contents, as well as those of the lateral sinus, if necessary.

TUMOURS OF THE BRAIN

Tumours are much more frequent in the brain than in the spinal cord; for the most part the same kinds are found in both organs.

The most frequent form of tumour of the brain is *sarcoma*. It often grows from the membranes, or commences in the skull and passes through the membranes to the brain. It may be a primary tumour, but is often secondary to growths in other parts of the body, and is then frequently multiple.

The next most frequent form of tumour is *glioma*. This consists of an overgrowth of the connective tissue, or neuroglia, of the brain. It may reach a diameter of two or three inches, is ovoid or globular in shape, and infiltrates the tissue of the brain in such a way that the divisions into gray and white matter are often perfectly apparent in their normal relations, though perhaps separated from one another, or, as it were, enlarged. The tumour is thus never encapsuled, but gradually shades off into the surrounding normal tissue. Under the microscope it is seen to consist of small cells, and of fine fibres forming a looser or denser network; and it can often be shown that the fibres are delicate processes connected with the cells. Sometimes the cells are of larger size, or the fibres less abundant, and thus the tumour comes more and more to resemble a sarcoma. The term *glio-sarcoma* is often used for these forms. Gliomata are very liable to hæmorrhage in their interior from rupture of vessels.

Tubercle occurs in two forms in connection with the brain: first,

in the form of minute tubercles in the membranes, in association with meningitis; secondly, as large masses in the cerebral substance, ranging from one third of an inch to two or three inches in diameter. These are globular in shape, consist of bright yellow caseous material bounded by an outer narrow zone of a pinkish-gray colour; while the adjacent brain-tissue is either normal, or slightly softened, with granule-corpuscles visible under the microscope. The tubercular masses can be easily detached from their bed. The gray edge of the tumour consists of cells resembling those of gray tubercle; the centre of the caseous portion may be softened into fluid or may be calcareous. These tubercular tumours are found in all parts of the brain, often in the cerebellum. They are sometimes multiple, and not infrequently accompany tubercular meningitis. They come in order of frequency next to glioma, and in children are proportionately more frequent.

Carcinoma is not very common: it is nearly always secondary. *Myxoma* and *fibroma* are rare.

Cysts occasionally occur, sometimes obviously as a result of a soft sarcomatous tumour breaking down in the centre, at others consisting of a simple membrane containing serous fluid, without any trace of a previous new growth. These have been found in the cerebellum.

Psammoma is a tumour, which grows from the membranes, and consists of fibrous tissue, with embedded particles of calcareous matter. *Cholesteatoma*, or pearl tumour, is another rare variety, which grows from the pia mater of the base of the brain; it is hard, shining like mother-of-pearl, non-vascular, and composed of horny epidermic cells arranged in concentric layers and enclosed in a fibrous capsule. *Lipoma*, *angioma*, and *melanoma* have also been observed.

Syphilitic Gumma.—This form of tumour grows commonly on the surface of the brain, and at first in the pia mater; it subsequently invades the substance of the brain, and contracts adhesions to the dura mater, so that the brain-substance and the two membranes are matted together. The tumour is irregular in shape, pinkish-red on section in the outer parts, yellowish and caseous in the centre. Sometimes syphilis causes a localised and diffused meningitis, without the formation of a definite gummatous tumour; and this is especially liable to occur at the base of the brain.

Parasites.—These rarely reach the cerebrum. A *hydatid* cyst (echinococcus) may grow in the hemisphere, and produce all the symptoms of tumour. The *cysticercus telæ cellulosæ* has been seen in the membranes and in the ventricles.

Ætiology.—This varies to a certain extent with the nature of the tumour. Some, as already stated, are secondary to new growths elsewhere. Tubercular tumours often occur in association with caseous changes in the bronchial or other glands. Gummata are rare in children, but frequent in adults, both early and late in the syphilitic history.

Males are more often the subject of cerebral tumour than females; and the most striking fact about the age is that tubercular tumours are much more common in children. Fagge noted that in tumours of the hemispheres the patients were mostly above thirty, while in tumours of the base they were mostly below that age. In a good many instances the symptoms have been attributed to a fall or a blow upon the head.

Local Effects.—In the immediate neighbourhood of a tumour the brain-substance is frequently affected, either by red or white softening, or by the yellow discoloration, which has been called yellow softening. If the tumour is large, the corresponding hemisphere of the brain is increased in size, and the convolutions are flattened. Even the cranial bones may be thinned by compression, so that they may be indented by the finger; this condition is known as *craniotabes*. Tumours in the middle lobe of the cerebellum or in the posterior lobes of the brain are frequently accompanied by ventricular distension or hydrocephalus, which is attributed to compression of the veins of Galen by the tumour.

Symptoms.—These are (1) *general*, and (2) *localising* or *focal*. The former are present in the majority of cases, and depend probably upon the increased intracranial pressure which any addition to the contents of the skull must cause; the latter are those which vary with the position of the tumour, and will help to show exactly where it is situate.

General Symptoms.—Headache is usually present, is often very severe indeed, and may be constant; but sometimes diminishes or disappears for a time. It may be felt all over the head, or it is limited to one region, and may serve as a localising symptom. Thus, there is generally occipital headache in cerebellar tumour, and, in a case under my care, a tumour of the right superior temporal convolution was accompanied by intense pain above the right ear. Headache is often absent with tumours of the motor area, or of the corpus callosum.

Vomiting is also a frequent and sometimes early symptom, and often leads to a wrong diagnosis of gastric disease. In its most characteristic form the food is regurgitated, without pain or effort or nausea, immediately it is put into the stomach; but nausea and straining sometimes occur in purely cerebral vomiting.

Optic neuritis, or inflammation of the optic disc, occurs in the majority of cases of cerebral tumour. It is almost constant in tumours of the corpora quadrigemina, exceedingly common in tumours of the cerebellum and posterior part of the cerebrum, but absent in more than half the cases of tumour of the corpus callosum, pons, or medulla. It is more frequently present when the tumour is a glioma or a cyst than when it is a tubercle (Martin, in *Lancet*, 1897, vol. ii.). With tumours of the cerebrum or cerebellum in patients under forty years of age it is rarely absent; but is more and more likely to be wanting as years increase (H. D. Singer). It is almost invariably double, but exceptionally

one eye has been alone affected; often one is more affected than the other, and the tumour is more likely to be on the side of the worse eye. After death the nerve has been found to present evidences of inflammation, and there is frequently an effusion of fluid within the sheath around the nerve.

Vision is at first but little affected, and even when optic neuritis is well marked, the defect of sight may be only discovered on careful examination.

After optic neuritis has existed some months, *atrophy* supervenes from contraction of the fibrous tissue of the nerve. Vision is now generally diminished, but is not always lost in proportion to the apparent atrophy; the pupils are generally dilated, and sluggish or inactive to light.

Convulsions are not so frequent as the symptoms already mentions, and they are very irregular in their occurrence in the cases attended by them; thus, there may be only two or three in the whole course of the illness, or they may be very frequent. Sometimes they are general and epileptiform in character; at others, they are limited to one or other region, and may then acquire a more localising value.

Among other general symptoms are mental changes; the patient may be dull, apathetic, forgetful, sleepy, careless, or untidy: in later stages he becomes lethargic, and finally comatose. Vertigo sometimes occurs. The pulse may be unusually slow. The nutrition may be unaffected, and the patient may even grow fat; more often, especially towards the end, flesh and strength are both lost. Constipation is common in cerebral tumour.

Localising or Focal Symptoms.—These may be absent, as in some tumours growing in the centrum ovale; but in most cases they are present, and generally the symptoms caused by a tumour of a particular region are in accordance with what has been determined experimentally as to the localisation of cerebral functions (*see* p. 320). It must be remembered, however, as already stated, that a tumour is not necessarily confined to one functional area, but may extend into, or press upon, others; and that slight pressure upon an area may stimulate the exercise of the function which greater pressure on, or destruction of, the area will abolish.

Tumours which compress *nerves* will cause a cessation of their functions—paralysis in the case of motor nerves, and anæsthesia in the case of sensory nerves (*see* Lesions of Cranial Nerves, p. 222, *et seq.*).

Tumours in the lower or outer parts of the hemispheres may implicate the *motor tract* and cause hemiplegia, which generally differs from the hemiplegia due to hæmorrhage or embolism in coming on quite gradually. Spasm, either tonic or clonic, also results from implication of the motor tract by tumours.

The distinguishing feature of tumours (and other local lesions) in the *cortical motor centres* is the occurrence of *Jacksonian epilepsy, i.e.*, localised convulsions (*monospasm*), or convulsions beginning

always in one part and unaccompanied at first with loss of consciousness. In these cases, according to the severity of the temporary disturbance in the part, there may be a twitching, say, of the hand alone, or of the hand and arm, or of all the muscles of the body; and when the convulsions are extensive, consciousness is generally lost. If they begin in the face, they spread successively to the upper limb, beginning with the fingers, and then to the lower limb: if they begin in the leg, the arm and face are successively affected: if in the arm, the face and the leg in turn. Bilaterally associated muscles (*see* p. 327) may be convulsed together. The convulsions are sometimes followed by temporary paralysis in the part in which the spasm begins. *Post-mortem* results have shown that convulsions beginning and spreading in the manner described are frequently due to a lesion in the part of the motor area corresponding to the limb which is first convulsed—namely, for the leg, the upper part; for the arm, the middle part; and for the face, the lower part of the central convolutions.

Tumours of the *frontal lobe* anterior to the motor area often cause dulness and stupidity, inability to fix the attention, loss of memory for recent events, and change of character: in addition to this, incontinence of urine (C. E. Beevor), impairment of smell, and fine tremor of the hand on the same side; but this is one of the regions in which a lesion may be entirely latent, and produce no symptoms.

Fits with a sensory aura of tingling and numbness have been observed in tumours of the *posterior central convolution* and *parietal lobe:* such growths may also cause loss of sensation in the opposite limb, affecting the distal more than the proximal parts.

Tumours of the *temporal lobe* in its posterior part may cause loss of hearing on the opposite side; in its anterior part, involving the uncinate region, they have diminished the sense of smell, or of taste, or have caused fits preceded by a disagreeable smell, or dreamy attacks, with visual hallucinations of persons and places, sometimes followed by convulsions.

Tumours of the *occipital lobe* situated in the cuneus cause hemianopia of the side opposite the lesion; *i.e.,* a tumour of the left occipital lobe paralyses the left retinal fields, and thus causes right half-blindness. If it extends forward to the angular gyrus, word-blindness will result, and if from its position or size it presses upon the cerebellum, ataxia, hypotonia, and nystagmus may occur.

Tumours of the *corpus striatum* and *optic thalamus* depend for their symptoms chiefly upon the implication of adjacent parts, *e.g.,* the internal capsule, by which paresis on the opposite side is produced. Pain, hemianæsthesia, and choreic movements on the opposite side are sometimes the result of a thalamic growth.

A tumour of the *corpora quadrigemina* causes reeling ataxy of the legs like that described below, with deviation to the side opposite the lesion. With this is associated double ophthalmoplegia, probably by pressure on adjacent parts (*see* p. 324).

A tumour of the *base of the brain* often involves the optic tracts or the nervi motores oculi: and thus may cause impairment of the movements of the eyeball, defective pupillary reactions, and failure of vision: together with some weakness of the muscles of the limbs on the opposite side. A tumour of the *pituitary body* may press upon both optic tracts, and thus cause temporal hemianopia: optic atrophy, drowsiness, and loss of the sexual functions, with tendency to grow fat, are also frequent. Acromegaly may be a consequence, but it is by no means constant.

If the *pons Varolii* is the seat of a tumour, any of the cranial nerves from the fifth to the eighth may be affected; and the motor tracts will be pressed upon, so as to cause bilateral, often unequal paresis, with spasticity and increased tendon reflexes. A completely unilateral tumour of its lower part may cause crossed hemiplegia (*see* p. 324).

Growths in the *cerebellum*, so long as they are confined to one or other hemisphere, may present no symptoms; but if of large size, they may extend forward so as to reach the pons, and involve the seventh nerve, or press downwards upon the pyramids of the medulla oblongata, and thus produce some weakness of the arm and leg on the opposite side. But a tumour in the middle lobe of the cerebellum produces some characteristic symptoms—headache aggravated by movement, ataxy, vertigo, oscillation of the eyeballs, and retraction of the head, or tetaniform convulsions. Ataxy and vertigo are the most common, and may be among the first symptoms (*see* p. 325). Optic neuritis is a constant accompaniment of tumours of the cerebellum.

The ataxy of cerebellar disease is different from that which forms the characteristic feature of tabes dorsalis or locomotor ataxy. The gait is reeling or staggering, like that of a drunken man; the patient sways from side to side, deviating generally to the side of the growth, readily overbalances himself, crosses his legs to regain his equilibrium, and often falls.

Experiments have shown a close relation between the parts of the cerebellum injured and movements of the body or eyes in particular directions; for instance, lesion of the anterior part of the middle lobe causes a tendency to fall forwards; lesion of the posterior part, a tendency to fall backwards; lesion of the middle peduncle causes lateral movements, or movements of rotation; and the eyes are moved upwards, downwards, to the right, or to the left, according to the different points selected for stimulation. But in slowly-growing tumours it is not always possible to recognise similar effects, as they are probably counteracted by cerebral influence; but if such symptoms occur, they may help to indicate the position of the lesion. Two other occasional effects of cerebellar tumour should be mentioned; one is a peculiar slow tremor of the muscles of the trunk and limbs, which has a certain resemblance to that of disseminated sclerosis; and the other, the globular enlargement of the head, which is due to ventricular distension, and is

therefore not peculiar to, but frequently occurs in, cerebellar tumour.

Duration.—Intracranial tumours usually last from six months to two years, and occasionally longer. Death takes place sometimes from exhaustion with vomiting, emaciation, and bed-sores; at others, from hæmorrhage into the tumour, if it be a glioma; at others, again, from intercurrent pneumonia. Tubercular tumours may end in a fatal tubercular meningitis. Not infrequently death takes place suddenly and quietly by failure of the respiration, while the heart continues to beat for some little time afterwards. In cases dying slowly the abdomen retracts, *tache cérébrale* may be obtained, and the breathing may acquire the Cheyne-Stokes character; the picture closely resembles that of meningitis.

Diagnosis.—The clinical symptoms above given will mostly serve to distinguish tumours of the brain. If headache and vomiting suggest gastric disease, the ophthalmoscope may prevent a serious error, or a careful examination may reveal uncertain gait or very slight paralysis. Double optic neuritis alone cannot now be regarded as conclusive evidence of a cerebral lesion, much less of tumour, as it may occur in anæmia, in meningitis, and in connection with disease of the ear; but it is of great value in association with other symptoms, and at least it excludes hæmorrhage and embolism, in which it very rarely occurs. The fact that vision often persists when optic neuritis is even well marked (*see* p. 213) renders it necessary that this lesion should be looked for, and an ophthalmoscopic examination should be made in any case where other symptoms suggest the possibility of cerebral tumour, even though the patient should state that he sees perfectly well. *Chronic meningitis* may be very difficult to distinguish from tumour, but *acute meningitis* is too rapid in its course, and is generally accompanied by fever; the long history will serve to distinguish the case of tumour in its last stages. *Renal disease* may simulate cerebral tumour in the headache, vomiting, and affection of sight; and even the ophthalmoscope may not at once clear up the case, since the optic disc is inflamed in albuminuric retinitis; and, on the other hand, optic neuritis from cerebral tumour is sometimes accompanied by retinal changes (brilliant white spots) precisely like those common in albuminuric retinitis. Albumin in the urine of course speaks for renal disease, but does not exclude a co-existing brain tumour; a constant localised pain, any local nerve-symptoms, and pronounced double optic neuritis, without further retinal changes, are in favour of cerebral tumour. As to the nature of the growth, the frequency of tuberculous tumours in children should be remembered, and the liability of gummata to occur on the surface. Cases with irregular paralysis of many cranial nerves are often syphilitic. Hughlings Jackson has said that convulsions beginning unilaterally, associated with double optic neuritis, are generally syphilitic. This is an expression of the fact that gumma may occur on the surface in the cortical area, and, like other causes of intracranial pressure, leads to optic neuritis.

Prognosis.—This is very unfavourable, unless the tumour can be removed by operation. Only syphilitic cases give any hope of improvement by drugs, and they frequently relapse again and again; they may indeed be fatal under the most vigorous treatment. It is probable that tubercles sometimes become arrested in their growth, or calcify.

Treatment.—In cases obviously syphilitic, potassium iodide should be given in doses of 10 to 30 grains three times daily, or even more, combined with mercurial inunction, and the internal use of mercury perchloride (60 to 80 minims of the liquor three times daily). Even in cases where a syphilitic origin is less certain, the iodide may be tried. Failing this, if the tumour can be certainly localised, its removal by operation should be considered. Many tumours have now been reached and removed, even in such deeply-seated parts as the pituitary body.

Besides the direct treatment of the tumour in cases suited for it, we may have to relieve symptoms. We may treat headaches by ice to the head, potassium bromide, or small doses of morphia; and sickness by effervescing salines, and tincture of iodine in 2 or 3 minim doses. The removal of a piece of bone by trephining will often relieve headache and vomiting, or check the progress of optic neuritis and delay or prevent the occurrence of amaurosis.

CHRONIC HYDROCEPHALUS

By hydrocephalus is meant the accumulation of fluid within the cranial cavity. An acute effusion is mostly determined by meningitis, either tubercular or posterior basal, and the former disease was once known as acute hydrocephalus.

A division has been made of chronic hydrocephalus into *internal* and *external* forms, according as the fluid is contained entirely in the ventricles of the brain, or is formed outside between the brain and the skull, in the subdural space. But much doubt exists as to the real occurrence of the latter class of cases, and their symptoms and general features are not materially different from the certainly more common cases of chronic internal hydrocephalus. It is true that in old age, and from other conditions, the convolutions of the brain diminish in size, the sulci widen, and the space in the skull thus left by the disappearance of brain-substance is filled up by fluid. Similarly, a loss by local shrinking of the brain is replaced by fluid on the surface. But this compensatory secretion has none of the effects of true hydrocephalus.

Hydrocephalus occurs in infants, and much more rarely in adults; the yielding nature of the cranial bones at the former age, and their solidity and resistance at the latter, cause some important differences in the course and symptoms.

HYDROCEPHALUS IN INFANTS

Ætiology.—Infantile hydrocephalus is either congenital, or is first noticed shortly after birth. Even if first observed later, its origin is usually quite obscure, but in some cases it has been due to posterior basal meningitis, by which one of the foramina has been obstructed, and the drainage from the ventricles has been impeded. When arising *in utero*, it has been attributed to blows or falls suffered by the mother. It has been observed to occur in several children of the same family. Tumours of the cerebrum or cerebellum, which cause hydrocephalus in adults (*see* p. 366), operate similarly in children, but produce greater enlargements of the head in proportion to the softer condition of the bones.

Anatomy.—In the obviously internal form of hydrocephalus, the ventricles of the brain contain an excess of fluid, sometimes amounting to a quart or more. It has the characters of cerebro-spinal fluid—that is, it has a specific gravity of 1006–1009, contains a small quantity of chloride of sodium, only a trace of albumin, and sometimes urea or cholesterin. The liquid may occupy all the ventricles, or all except the fourth, or the two lateral ventricles alone. By its increasing quantity the substance of the brain is enormously distended, the convolutions are flattened, sometimes reduced to a few lines in thickness, and the basal ganglia are correspondingly thinned out. The aqueductus Sylvii may be distended to the size of the finger when the fluid is in the fourth ventricle; it is often closed when the fourth ventricle is not dilated. In extreme cases the distinction between gray and white matter is lost in the parts exposed to most pressure; the ependyma is often thickened, and contains amyloid bodies, while its surface is covered with fine granulations.

Hydrocephalus is sometimes associated with other lesions of the central nervous system, *e.g.*, spina bifida or syringomyelia.

Symptoms.—The most obvious, and it may be for a time the only, symptom of the disease is the condition of the child's head which results from it. The pressure on the brain is transmitted to the skull, and as this expands outward the head becomes enlarged. The enlargement is often extreme. In congenital cases it may form a serious obstruction to delivery; in others it appears in the first few months of life, and the circumference may amount to twenty-four or even thirty-two inches, instead of sixteen or eighteen inches, which are the usual measurements up to the age of one year. The head is at the same time globular, and the skull projects over the face and neck almost uniformly all round. The face looks small and shrunken in proportion, and has a distressed, anxious, or senile expression in severe cases. The distension from within drives the orbital plates outwards, and the eyeballs are turned down so that the lower part of the iris and of the cornea is lost under the lower eyelid,

and the upper part of the sclerotic is exposed. The increased size of the head is due to a separation of the cranial bones from one another, so that the fontanelles are much enlarged and the sutures widened. In these spaces fluctuation can sometimes be felt. In cases of long standing it is found that ossification has gone on at the margins of the bones, advancing into the sutures, so that ultimately, if the patient lives, by this means, and by the formation, from independent centres in the membrane, of fresh plates of bone (*ossa triquetra*, Wormian bones), the deficiencies of the skull may be completely filled in. In the early stages, however, the bones are thin, wanting in diploë, and transparent. The skin of the scalp is tightly stretched, excessively thin, and large blue veins ramify over the surface. The hair is generally scanty. In some cases, where the fluid is not very abundant, the bones may yield sufficiently to obviate any considerable pressure upon the brain-substance. The symptoms may not then go much beyond the enlargement of the head; at most there is some general weakness and loss of flesh, from which after a time the child recovers. But in most cases there are other symptoms. The muscular power is deficient; especially the large head cannot be held upright, and falls from side to side, or has to be supported by the hands when the child sits up in bed. The child cannot walk, or acquires the art in moderate cases only after a long time. Vision is often defective or lost; and in extreme instances there is atrophy of the optic nerves, which has been preceded in some cases, it appears, by optic neuritis. The other senses may be, to a certain extent, impaired. The intellectual functions are often defective. The child slowly learns to talk, continues childish out of proportion to its growth, and is fretful, irritable, or vicious in temper. Nystagmus, rigidity and spasms of the weakened limbs, convulsions, and vomiting occur often in severe cases. Many of these patients die young, relapsing into a condition of apathy or semi-coma, lying in bed with eyes closed or twitching, with rigid limbs, and incontinence of urine and fæces, constantly moaning or whining, and refusing food or else eating voraciously.

Finally, convulsions, or coma, or some intercurrent disease, such as bronchitis, pneumonia, or measles, may end the scene.

In some cases the fluid has escaped by rupture of the integuments or by bursting through the nose or eyes.

The duration is variable. In the mildest cases recovery may take place, or rather the disease is arrested; other patients live to four, five, or six years, or more. I have recorded a case ("Clin. Trans." 1897) where a lad reached the age of sixteen with perfect mental development and physical capacity; and died then with rapid cerebro-spinal symptoms, the ventricles containing thirty ounces of fluid. A few cases have lived to sixty or seventy.

Diagnosis.—Confusion is most likely to take place between this and rickets. The rickety head is cubical in form rather than spherical, the vertex being flattened; the downward displacement

of the eyeballs is absent; the limbs may be feeble, but the mental powers are not deficient; and the other evidences of rickets—beaded ribs, thickened wrists, sweating of the head, and general tenderness—are present at one time or another.

Treatment.—This is not very promising. Perchloride of mercury and iodide of potassium have been given with no appreciable effect. Mechanical treatment by pressure, or the removal of fluid, or both combined, is often useless, and is not free from danger of hastening the end.

Pressure is best applied by covering the head with narrow strips of adhesive plaster carried from front to back, with a long strip laid two or three times round the circumference of the head, so as to fix the whole firmly. After ten or fourteen days fresh strapping may be applied. If fluid be removed, the amount should not exceed two or three ounces, and the trocar may be inserted at the outer angle of the anterior fontanelle; the head should then be strapped. If no harm results a similar quantity may be again withdrawn after two or three weeks. The fluid may be also drained off by lumbar puncture.

HYDROCEPHALUS IN ADULTS

Ventricular distension in adults arises from (1) mechanical interference with the circulation of the brain; (2) meningitis or meningo-ependymitis.

1. *Interference with the Cerebral Circulation.*—The larger proportion of these cases probably occurs after the period of life at which any very great expansion of the head can take place. The head nevertheless becomes globular and somewhat enlarged. This is often seen in cases of cerebellar tubercle, or other tumour situate there or in the posterior lobes of the brain, whereby the veins of Galen or the straight sinus may be compressed. The return of blood is thus delayed, and effusion into the ventricles is the consequence. It is not generally possible to distinguish the symptoms due to the hydrocephalus from those of the tumour which causes it.

2. *Meningitis* and *Meningo-Ependymitis.*—Some of the cases of chronic hydrocephalus in adults, which are not the result of the growth of tumour, show after death evidences of an inflammatory origin, such as thickening and granular condition of the ependyma, cell-infiltration of the sub-ependymal tissues, thickening, opacity, or matting together of the membranes at the base, or adhesion of the membranes to the brain. The fluid reaches a much smaller amount than in the infantile cases; the convolutions are more or less flattened; the bones are thin, and wanting in diploë. Sometimes the sutures have opened again. Epidemic cerebro-spinal meningitis is an occasional cause of hydrocephalus.

Symptoms.—These are not, as a rule, distinguishable from those of some chronic disease of the brain, such as tumour, and chiefly because the enlargement of the head, due to the expansion of the bones of the skull, is absent. The symptoms that have been

noticed are pains in the head, vomiting, numbness in the feet and legs, weakness or paralysis of the limbs, sometimes even hemiplegia, though the lesion is bilateral, blindness with optic neuritis or atrophy, delirium, strangeness of manner, stupor, convulsions, and coma. The symptoms may be aggravated by exercise or the use of alcohol. Death may be gradual or sudden.

Treatment.—In cases in which the diagnosis can be reasonably made, the use of perchloride of mercury, iodide and bromide of potassium, and local counter-irritation or anodyne applications for the relief of pain, are indicated. In the acuter cases lumbar puncture may render good service.

GENERAL PARALYSIS OF THE INSANE

(*Paralytic Dementia*)

Although mental diseases do not come within the scope of this work, it is desirable to describe this complaint, because it depends upon actual structural changes in the central nervous system, and because the paralytic symptoms are often the prominent features of the case for long periods of time, and may give rise to a difficulty in diagnosis from other purely physical conditions.

Shortly stated, the disease consists in progressive symptoms, partly of a paralytic, partly of a mental character, terminating in dementia and complete loss of power, and dependent upon widely spread anatomical changes in the brain, spinal cord, and nerves.

Ætiology.—With regard to its causation, general paralysis of the insane stands in the same position as tabes dorsalis, and is similarly regarded as a parasyphilitic disease. Syphilis is an antecedent in about three-quarters of the cases, and when the paralysis occurs in children or quite young persons, there has been syphilis in a parent. In 95 per cent. of the cases the Wassermann reaction can be obtained from the cerebro-spinal fluid ; and in all cases from the blood-serum. Cases have in the past been attributed to other factors, such as sexual excess, alcoholic indulgence, mental worry and overstrain, business anxieties, and injury : but their influence can only be of secondary importance.

The disease is much more frequent in men than in women, and occurs mostly between the ages of thirty and fifty ; in other words, the symptoms rarely appear in less than ten years after infection.

Symptoms.—Considerable differences are seen in the grouping of the symptoms ; in some cases the paralytic features are more prominent, in others the mental. The paralytic symptoms are sometimes cerebral in origin, at others spinal, and they may for a time exactly resemble those of tabes dorsalis or disseminated sclerosis ; the mental failure may be at one time marked by exaltation, at another by depression, but ultimately, in all cases, dementia supervenes. In that which has long been considered the most typical form of general paralysis of the insane, the first thing noticed

is often an alteration of a moral or intellectual kind; and this may be for some months or a year or two before anything more decided is observed. The man becomes careless or neglectful, tends to intemperance in drink, or spends money more freely than has been his wont, without any justification; or he is irritable or restless, changing in his affection to his wife or family, or jealous without cause. The first indications on the physical side are generally tremor of the tongue or lips, or hands; the gait then becomes uncertain or tottering, but without any well-characterised ataxy. More marked weakness of the tongue and lips causes defects of articulation. Many-syllabled words, such as "artillery" or "biblical," are confused, or some syllables are doubled or misplaced; other words are clipped short. The handwriting is uncertain and shaky, and letters are apt to be dropped out, or words left unfinished; and in turn all other finer movements, which have been attained by careful education, such as piano-playing and violin-playing, are lost. The reflexes are exaggerated. The pupils are often unequal, and sometimes closely contracted; but the Argyll-Robertson symptom is not generally present. By the time that these forms of weakness have become pronounced, if not before, the mental deficiency has reached the stage of delusions, and in many cases these delusions are of an exalted kind, and ideas of grandeur fill the mind of the sufferer (*megalomania*). These concern himself alone; they express what he is, what he possesses, or what he can do. He is the Almighty, the King of England, or the Prime Minister; the most handsome, or the most powerful man in the world. He has boundless wealth, hundreds of carriages, millions of gold watches, or countless wives. Sometimes the first indication of these ideas is revealed by his going to a shop and ordering useless quantities of expensive goods, far beyond his means. Restlessness of body and mind is also a characteristic at this stage.

There is then either a gradual transition to a *second stage*, or a more rapid change by one or more convulsions, from which the patient recovers considerably worse that he was before. His mental power is more deficient, his memory fails him; the lofty ideas may be present, but he is less influenced by them, and is more manageable. The muscular weakness is more apparent: it shows itself in the want of expression in the face, in the very imperfect articulation, and in loss of power in the arms and legs. Common sensibility is also diminished or lost; but appetite is often retained, and the patient is not infrequently fat.

He gradually becomes more feeble-minded, and, finally, the *third stage* of complete dementia, with loss of control of the bladder and rectum, is reached. He sits about, or is too weak to do more than lie in bed; he is subject to convulsions from time to time; the limbs may become contracted; and bed-sores will form unless the greatest care be taken. He dies from pneumonia or bronchitis; or swallowing is difficult, and he may be choked; or cystitis or bed-sores may lead to secondary blood-poisoning.

Varieties.—Some cases never show the stage of exalted ideas, but are *melancholic* from the first, and gradually pass into the final condition of dementia; others are *demented* throughout, without exaltation or melancholia. A *double* form is also spoken of, in which the stage of exalted ideas is followed by one of depression, and this again by one of exaltation. The early paralytic symptoms are sometimes distinctly spinal. The case presents, perhaps for some years, all the characteristic features of *tabes dorsalis*, with shooting pains, absent knee-jerks, locomotor ataxy, and Argyll-Robertson pupils; or of *lateral sclerosis*, with weakness, rigidity, and increased reflexes. Savage says this is not infrequent in women, single as well as married, and at a rather younger age than is common with other varieties. Or the features are those of *disseminated sclerosis*, with nystagmus, staccato speech, and oscillations of the limbs and trunk on movement; or *mixed* forms, suggestive partly of one, partly of another, of these three diseases, may be present.

Some special conditions of the nervous system and nutrition may be mentioned. Fits or convulsions may occur at almost any period of the case; they may be slight or severe, passing off very quickly or leaving the patient comatose for some time; the convulsions also may be scarcely noticeable, or unilateral, or general. Amongst the ocular symptoms the pupils have been already mentioned; but the optic discs are often unaffected, and ptosis and strabismus are rare. The muscles seem to present no constant changes either in nutrition or in electrical reactions. The bones are brittle; the skin is often pale, waxy, and especially greasy; sometimes bullæ form, and a marked capillary congestion over the malar bones is common. There is a tendency to whitlows on the fingers, and to subcutaneous hæmorrhages, which sometimes lead to septicæmia.

The temperature is high in acute cases, and after convulsions, when it is often accompanied by free sweating. It is also raised by much bodily exertion or mental excitement, and by complications such as bed-sores and lung disease. The cerebro-spinal fluid obtained by lumbar puncture contains lymphocytes in excess.

Duration.—This is variable; there are acute and chronic cases. If the early symptoms of gradual mental change, and the early spinal symptoms, in cases beginning with locomotor ataxy or sclerosis, be excluded, the duration is not often more than two years.

Morbid Anatomy.—The lesions are very variable, but the following are found in different cases: Thickening of the calvarium, which is much marked by the Pacchionian bodies; thickening of the dura mater, with false membranes (pachymeningitis); abundant subarachnoid fluid, with thickened or adherent membranes, the adhesion, when present, being more over the frontal, parietal, and temporo-sphenoidal lobes, and more on the upper than the lower surface; wasting of the convolutions, especially the ascending parietal, paracentral, and first frontal at its base; a violet-red colour of the cortex of the brain; in some cases much fluid in the

lateral ventricles, with softening of the brain-tissue; in a larger number, a general hardening of the brain. In the spinal canal the same changes may be found; pachymeningitis, or adhesion of membranes, or effusion of blood within the dura mater. The spinal cord is wasted, or presents the lesions of posterior or of lateral sclerosis. Microscopic examination shows the following changes: The pia mater is infiltrated with lymphocytes, plasma-cells and mast-cells. The brain shows proliferation of the neuroglia especially in the superficial layers of the cortex, with large glia cells, new-formed blood-vessels, thickening of the intima and adventitia of the vessels and infiltration of their lymph-sheaths with lymphocytes and plasma-cells, destruction of the myelin sheaths of the nerve-fibres, and degeneration and sclerosis of the ganglion cells, especially of the pyramidal cells of the third layer. The sympathetic ganglia are, according to Savage, not appreciably affected. The disease is thus a chronic meningo-encephalitis with increase of connective tissue and degeneration of the neurons: the origin in syphilis, the association with the spinal sclerosis, and other facts, make it highly probable that the degeneration of the nerve-elements is primary (Mott).

Diagnosis.—*Alcoholism* may be mistaken for general paralysis, the tremor of the lips, tongue, and hands largely contributing to this; commencing *peripheral neuritis* might further complicate the case. But the close association of the symptoms with continued drinking, the absence of inequality of the pupils, and the improvement on prolonged abstinence, would point to alcoholism. Mental failure, with definite cerebral lesions such as *tumours*, or the dementia following *apoplexy*, may give rise to difficulties. From the general physician's point of view, it is important to recognise that various anomalous paralytic symptoms may be the first symptoms of general paralysis. If a case is typically tabes dorsalis there is no special reason to anticipate mental trouble; but if the symptoms develop very rapidly or present unusual groupings, or if there are mixed symptoms not conforming to the ordinary types of the spinal-cord diseases, the mental condition should be closely scrutinised, and the possibility of general paralysis of the insane should be kept in view.

The diagnosis of general paralysis may be much assisted by an examination of the cerebro-spinal fluid obtained by lumbar puncture. This fluid shows three changes. (1) It contains the anti-body shown by the Wassermann reaction. (2) It contains an excess of proteid, to the extent of four or five times the normal: this proteid is partly albumin, but much more largely euglobulin. A characteristic test for this is that of *Noguchi*. Half a cubic centimetre of a 10 per cent. solution of butyric acid in physiological saline is added to ·1 c.c. of clear cerebro-spinal fluid, and the mixture is heated to boiling point. To this is then added ·1 c.c. of a 4 per cent. solution of sodium hydrate and the mixture is again heated. It then becomes cloudy, and peculiar light gray flocculi form in from two to thirty minutes.

(3) The lymphocytes, which normally do not exceed 5 per cubic millimetre, are increased to some figure between 100 and 1000.

Dr. Mott points out that in early cases of neurosyphilis, which may yield to anti-syphilitic treatment, the blood-serum will probably give a positive *antigen* reaction, while the cerebro-spinal fluid may show neither *antigen,* nor anti-body: in the later and more hopeless parasyphilis both blood-serum and cerebro-spinal fluid show the presence of anti-body.

Prognosis.—In an undoubted case it is bad, death being the certain termination; but temporary improvement sometimes takes placé, especially in the cases with exaltation of ideas.

Treatment.—This is, of course, in the highest degree unsatisfactory, as nothing seems able to stop the progress of the very widespread changes in the nerve tissues. If the disease is recognised early, the patient should be at once removed from all sources of worry, anxiety of business, &c.; he should get change of scene, but should be kept under observation. Excess of every kind is to be avoided; and hitherto drugs have been found to be of little or no service except in ameliorating symptoms. Antisyphilitic remedies have not so far been very successful in this or in the analogous case of tabes dorsalis; but in early cases, and especially when the original infection was inefficiently treated, mercurial inunction or other treatment for syphilis should be fully tried.

DISEASES OF THE SYMPATHETIC NERVOUS SYSTEM

Morbid histological changes, such as atrophy, pigmentary or fatty degeneration, fibrosis and hæmorrhage, have been found in the ganglia of the sympathetic system, but little appears to be known of a definite association between such changes and functional disturbances or symptoms as a consequence.

The best illustration of a direct interference with the sympathetic nervous system is given by those cases in which the sympathetic cord in the neck has been pressed upon, cut or otherwise injured, as, for instance, by stabs, gunshot wounds, operations on the neck, and the pressure of tumours, enlarged thyroid gland, or the extension of pleurisy of the apex of the lung. The results are generally paralytic in kind, and are the following: contraction of the pupil on the same side; narrowing of the palpebral fissure; later, or less constant, withdrawal of the eyeball within the orbit (*enophthalmos*), attributed to shrinking of the orbital fat, and paralysis of the smooth muscular fibre in the orbit; dilatation of the vessels of the head and neck on the same side; increase of temperature on the same side; alteration of the sweat secretion on the same side, sometimes diminution, at others an increase: exceptionally, thinning of the face, or graying of the hair on the same side.

Occasionally the sympathetic cord seems to be irritated rather than injured, and the symptoms are the converse of the above, namely dilated pupil, prominence of the eyeball, contraction of the vessels with pallor of the skin; but less is known of the structural pathology which will cause them. It must not be forgotten that such sympathetic symptoms may arise from lesions in the cilio-spinal region of the spinal cord, or in the brain, and may be functional or reflex. Not much definite is known of organic lesions of the thoracic and abdominal sympathetic and the symptoms referable to them, though it may be supposed that disturbances of the thyroid gland, of the heart's action, of the action of the intestines, and perhaps abdominal pains, would be among the symptoms.

Functional disorders of the sympathetic system are not uncommon, and especially for this reason, that the vasomotor apparatus is controlled by the sympathetic; and a number of conditions characterised by more or less persistent vascular disturbances are probably referable, directly or indirectly, to sympathetic disorder. Such are persistent flushing, throbbing of vessels, irregular sweating, many symptoms in neurasthenia and hysteria, the climacteric phenomena,

angio-neurotic œdema, erythromelalgia and acropathy in general, intermittent claudication, Raynaud's disease, exophthalmic goître, myxœdema, and progressive facial hemiatrophy. Most of these disorders are dealt with in other parts of this book.

Progressive facial hemiatrophy, a rare and curious affection, begins usually between the ages of ten and twenty, without, as a rule, any definite cause. There is a gradual atrophy of one side of the face and head, involving the skin, subcutaneous tissue, bones and muscles, so that a remarkable unilateral deformity is produced. The muscles of the jaw and tongue may participate, but it is not a degenerative atrophy. The lesion is progressive, but ceases sooner or later. It is often associated with other neuroses, but there is still much doubt about its actual cause.

FUNCTIONAL DISEASES OF THE NERVOUS SYSTEM

EPILEPSY

Epilepsy is a disease in which there are attacks of sudden loss of consciousness with or without convulsions, independent, as far as our present knowledge goes, of any demonstrable lesion of the brain, or peripheral irritation, or blood-poisoning. Although the name is commonly associated with the idea of convulsions, and these indeed occur in the most typical and severe forms of attack, still it is important to note that coma is almost invariably present with the convulsions, and in many slighter attacks there is no convulsion at all. The second part of the definition excludes those convulsions which may arise from organic cerebral lesions, such as tumour, or from Bright's disease, or from anæmia. These are often called *epileptiform* to indicate their close relation to the *epileptic* convulsions now under consideration.

Epilepsy, then, so far as the brain is concerned, is a functional disorder, a neurosis, or spasmodic neurosis, as it has been named by some; and its recognition, to a certain extent, depends on the absence of any other symptom from which the existence of structural lesions or diseases likely to cause convulsive phenomena could be inferred.

Ætiology.—It is slightly more frequent in females than in males, and very much more often begins in early life (Gowers says 75 per cent.) than in the middle or advanced age, though it may be common enough in the latter class, simply because it is in a great majority of cases incurable, yet not fatal. Thus, beginning

at an early age, it may continue throughout the life of the individual, who eventually dies from other causes. Among the predisposing conditions, inheritance has the greatest importance; and this shows itself partly by the appearance of epilepsy in the children of epileptics, but very largely by its occurrence in the offspring or other blood relatives of those who have suffered, not from epilepsy, but from some other serious disorder of the nervous system, such as insanity, hypochondriasis, hysteria, and marked neurotic weakness, or nervousness. Dipsomania in the parents may occasionally be a contributing cause. When epilepsy is not inherited but acquired, alcoholic indulgence, sexual excesses, and possibly masturbation, may be the predisposing causes. But the first two are not so likely to be in operation at the period of life when epilepsy generally begins, and the last more often leads to hysterical conditions simulating epilepsy—hysteroid epilepsy. The more immediate causes of a first epileptic attack, which may be the beginning of a lifelong series, are fright, mental anxiety or excitement, injuries to the head, fevers such as scarlet or enteric, and the presence of worms in the intestines.

Epilepsy occurs in two well-marked forms, described as major and minor attacks. The first is a fully-developed fit with coma and convulsions. The second is a momentary loss of consciousness, with little or no convulsion; or, rarely, slight motor disturbance without unconsciousness.

Major Epilepsy or Haut Mal.—This occurs in several stages—(1) aura; (2) unconsciousness and tonic contraction; (3) clonic convulsion; (4) recovery. The *aura* (or breath, from the sensation of air passing up the limb to the head, which is one form of this symptom) is any sensation or motion experienced by the patient while he is yet conscious, mostly of very short duration, and terminating abruptly in loss of consciousness and convulsion.

(1) There is a great variety of auræ, which may be felt in almost every part of the body—in the limbs, face, and head, in the viscera, and the organs of the special senses. They have been classified as sensory, motor, secretory, vasomotor, and psychical. The following may be mentioned: tingling and numbness in the arm, leg, face, or tongue; twitchings or spasms in the same parts; loss of vision, or visual hallucinations, such as flashes of light, or colours (generally red or blue) or definite objects or enlargement of surrounding objects (*megalopsia*); hallucinations of sound, noises, &c.; unpleasant odours or tastes; sensation of choking, nausea, vertigo, epigastric pain; flushes of heat, coldness, perspiration, palpitation of the heart; an indefinite sense of fear or anxiety; running or jumping, or other co-ordinated movement. Auræ of sensation and motion are mostly unilateral, but may be bilateral; the arm is more often affected than the leg, and facial auræ mostly consist of spasm. Visual auræ are much more common than auræ of the other special senses. Sometimes a vague sense of fear may last some time before the occurrence of the actual fit; but, as a rule,

the aura is of momentary duration. In about half the cases it is entirely absent.

(2) The fit itself commences with sudden unconsciousness; if standing or walking, the patient often falls suddenly forwards, or seems to be thrown violently to the ground, sometimes with an involuntary cry, shriek, or low tremulous groan—the epileptic cry. He is then found to be in a state of tonic convulsion, the back rigid and slightly arched, the legs extended, and the head drawn backwards or rotated to one side. The face is often pale at first; the pulse is quick, but sometimes it cannot be felt, and this has been attributed to compression of the artery by muscular contraction; the pulse has also been observed to cease at the moment of unconsciousness. The general tonic contraction fixes the chest, and respiration is stopped, so that the face becomes more and more dusky, and eventually is quite cyanosed. The tonic stage lasts from three to thirty or forty seconds, and then passes into the stage of clonic convulsions.

(3) Twitchings begin in the face, the eyelids, and the side of the neck, and quickly extend to all the muscles of the body and limbs. There is a rapid succession of to-and-fro movements, of alternate flexion and extension in the limbs, of opening and shutting of the eyelids and of the jaws, lateral deviation of the eyeballs, and perhaps of the head; the tongue is pushed forward, and may be caught between the teeth; saliva is freely secreted, frothed in the mouth, and escapes from the lips mixed with blood from the bitten tongue. The face becomes livid, or almost black, and the lips and features are swollen. Urine, fæces, and in men, semen may escape during this stage, and the violent contraction of the muscles may even cause dislocation of the shoulder. The patient is, of course, quite insensible; the conjunctivæ do not respond to a touch, and the pupils are dilated or oscillate.

(4) The clonic stage lasts a few minutes, rarely more than five or six, and then the convulsions gradually subside—they become less frequent, and are interrupted by pauses of some seconds; the breathing becomes easier, the frothing at the mouth ceases, and the face gradually assumes a more normal colour. Finally, the patient remains simply comatose, and the coma passes into natural sleep, or consciousness is recovered rather suddenly soon after the cessation of the convulsions.

The reflexes are mostly absent for a short time after the attack, and then for a time the deep reflexes may be increased. The plantar reflex after a temporary absence is at first extensor, then again normally flexor. The urine may contain a trace of albumin or sugar; petechiæ may be seen under the skin from rupture of blood-vessels during the stage of venous congestion; sometimes there is a transient hemiplegia; or vomiting; or serious mental disturbances, such as delirium, which is often of a maniacal kind.

The mechanical injuries from which the epileptic suffers will, of course, remain after the fit, and may give valuable indications

in cases where the fit has not been seen—for instance, in nocturnal epilepsy. These are the bitten tongue, petechiæ on the skin, a dislocated shoulder, and, in other cases, various cuts, wounds, or bruises, from the falling of the patient upon the ground or against unyielding objects.

Minor Epilepsy or Petit Mal.—This consists, in a large number of instances, of little more than a sudden unconsciousness; in the midst of talking, perhaps, the eyes become fixed, the pupils dilated, the speech incoherent, and the patient is obviously unconscious of what is going on around him; he may, if at meals, put his fingers in his plate or his cup, or commit some other irregularity that he would not do if conscious. The condition lasts a few seconds, and then he becomes conscious, and goes on with what he was doing, or perhaps recognises that there has been a blank, or feels giddy, or has headache, and is glad to lie down for some time. Sometimes giddiness is the most marked feature of the attack, and in other cases a sensation in one or other part of the body, or a spasmodic movement, which may be quickly followed by temporary unconsciousness, though the former will seem to the patient the chief feature of the attack. These have a close resemblance to the auræ of the major attacks, and include sensations in the epigastrium, hands, head, nose, eyeballs, and cardiac region; olfactory and visual sensations; jerks in the limbs, head, or trunk; sudden tremor, screaming, or dyspnœa; mental conditions, such as a sudden state of fear, &c.

Post-epileptic Conditions.—The *petit mal*, even more than the major attack, is liable to be followed by certain post-epileptic conditions. One of these consists of various automatic actions, of which the patient is then and afterwards entirely unconscious. He may thus commit acts of violence, rushing about and striking all that he comes near, or a woman may kill her child, or one may appropriate things that do not belong to him. Trousseau records the case of the judge who relieved his bladder in the corner of the room without any consciousness of the act. These cases have great medico-legal importance, since the occurrence of epileptic fits may be quite unknown, and the criminal acts may be attributed to wilful and conscious violence. Sometimes these attacks are maniacal in their character (epileptic mania), and the automatic actions are accompanied with much mental disturbance, such as terror, violent passion, and hallucinations. In girls, boys, and young women, the minor attack may pass into a hysteroid condition (*see* Hysteria).

Though we can generally distinguish between the major and minor attacks, there are attacks which present intermediate characters. The two forms are often only different phases of the disease in the same person; thus it is not uncommon for children to suffer first from minor epilepsy, and as they get older to develop the major attacks. They may both occur in the same patient alternately, or more or less irregularly.

Course of the Disease.—The frequency of epileptic attacks

varies considerably in different cases, and at different periods in the same cases. Thus, there is generally an interval of one or more months between the first and second attacks, but with the progress of the disease the intervals often become shorter, and the fits may be as frequent as two or three in a week, or even several in a day. In some cases two or three fits occur in quick succession, or at short intervals, and the patient is then spared for a long time. A severe fit is much more likely than a slight one to be followed by a long interval. Probably alcoholic indulgence, injudicious feeding, and mental or physical over-exertion, increase the frequency of the fits. In some female epileptics the attacks come on with each menstrual period.

In rare instances the patient has a series of fits, extending over some hours, or one or two days, and never recovers consciousness in the intervals between them. The heart beats violently and rapidly, the respirations are quick, twitchings occur in the intervals of the convulsions, the temperature often rises to 105° or 107°, and the patient may die collapsed, or may become delirious. This is called the *status epilepticus;* it is often fatal.

Health between the attacks.—This depends a good deal upon the frequency of the fits. Where these are not numerous, the individual may enjoy excellent health. Many epileptics are strong, hearty, and vigorous, never ailing at all except at the time of the attack. When, however, the fits are very frequent, or the disease has lasted a long time, the mind generally suffers, the patient becomes dull and irritable, the memory is deficient, and intellectual processes are slower; and in some cases actual imbecility is the result. In children, sometimes, even after a few fits, permanent imbecility or mania may be developed.

Death from epilepsy is by no means common, and, except in the case of the rare status epilepticus above described, it is mostly the result of some injury to which the patient is exposed during the fit. Thus, during a fit he may be thrown from a height, or fall into water and be drowned, or be choked by food, or he may be smothered in bed by his face being buried in the pillow, or he may die later from injuries received by a fall into the fire, or from a carriage or bicycle.

Pathology.—In fatal cases of old standing some thickening of the bones of the skull or of the cerebral meninges has been found; and more recently sclerotic changes in the cortex, and degeneration of the cortical cells, with chromatolysis and vacuolation, have been found. Some of these may be of a secondary nature, but there is an increasing tendency to believe that structural changes may after all be the real cause of the disease. A theory of *auto-intoxication* has been based upon the fact that the urine contains more toxic products after the fit than at other times. The viscera are congested in those who die in the actual fit. A. E. Russell believes that a sudden failure of the cerebral circulation is the immediate cause of epileptic (and many other) convulsions.

That the cause of the epileptic fits has its seat in the cortex of the brain is shown by the following facts: The association of coma with the convulsions; the existence of cases of masked epilepsy, in which the symptoms are mainly psychical, such as epileptic mania and delirium; the fact that definite lesions of the cortex, such as tumour or gumma, produce convulsions identical in character with those of epilepsy—that is, the close resemblance between the convulsions of true epilepsy and Jacksonian epilepsy (*see* p. 368); the generally uniform spread of the convulsions from face to arm, and arm to leg, corresponding to the relative positions of the motor areas for those parts on the surface of the brain—a result which may be observed not only in the idiopathic epilepsy of man, but in the epileptic convulsions produced by experimental irritation of the brain in animals; the origin of some cases of epilepsy in definite lesions of the cerebral cortex—*e.g.*, as the result of blows; the frequency of epileptic convulsions in congenital or infantile cerebral lesions—*e.g.*, cerebral diplegia; and lastly, the cases occasionally recorded in which epileptic fits have ceased after the development of disease in the internal capsule.

Diagnosis.—Epilepsy is with no great difficulty recognised when actually seen, but one is often called upon to prescribe for fits which only occur at times when the physician cannot witness them; and it is not always easy to come to a right conclusion from the descriptions of friends. The major attacks have to be distinguished from attacks of hysteria, and from simulated fits; minor epilepsy from attacks of syncope. In *hysterical attacks* the movements are more purposive, or more clearly the result of external stimuli; they are not mere alternating contractions and relaxations of antagonistic muscles, but more combined movements, apparently made with an object. Thus, the patient may dash her head repeatedly against the floor or the bed; and, if efforts are made to restrain her, she will struggle to throw off those who are holding her, or will bite and clutch those near her. The facial muscles may twitch, and some saliva may come from the mouth, but it is not tinged with blood, and the tongue is not bitten. The face is generally red or pale, sometimes rather blue about the lips, but it never presents the intense cyanosis of epilepsy. The eyelids often quiver, and resist attempts to separate them. The fit of hysteria is of long duration, lasting half an hour or longer, whereas that of epilepsy is over in a few minutes. The mere fact of unconsciousness is not conclusive, as the events of a hysterical fit are not in the least recalled by the patient. But in hysteria there is an automatic response to sensory and auditory impulses, while in epilepsy the patient is, for the time, absolutely senseless.

In *Jacksonian epilepsy* the unilateral localised convulsion is primary, and loss of consciousness is either absent or secondary.

The *malingerer*, who attempts to excite sympathy as a sufferer from epilepsy, can, with a little care, generally be detected. He is careful to fall so as not to hurt himself, whereas the epileptic is

thrown down suddenly, and if in the street will probably strike his head or face, or will fall in the road, not making any effort to save himself. The malingerer is red in the face, rather than pale or livid; his skin perspires from the exertion, his pupils are not dilated, and are sensible to light. The fact that he has not lost consciousness may be tested in various ways: by touching the conjunctiva, when the eyelids will close, though he will probably resist attempts to raise the upper eyelid; by applying snuff to the nostrils; by producing some very painful impression, as by forcing one's thumbnail under that of the malingerer.

Minor epilepsy, or *petit mal*, is distinguished from *cardiac syncope*, or simple fainting, by its occurrence under circumstances not conducive to fainting, by its suddenness, and by its rapid recovery, followed by mental confusion rather than physical prostration. The occurrence of spasm or of any warning sensation other than the feeling of faintness is in favour of epilepsy. Syncope comes on more slowly, and is recognised as a gradually increasing faintness by the patient. Still, this may happen as a warning sensation of *petit mal*. To distinguish other forms of *giddiness* from the vertiginous form of minor epilepsy, one must remember that ordinary vertigo is not accompanied by loss of consciousness, and that in the aural form, or Menière's disease, there are persistent deafness and tinnitus.

When it has become certain that, in any case, the convulsions are really epileptiform in character, it has yet to be determined that they are not due to tumour of the brain, peripheral irritation, or the uræmia of Bright's disease, before we can pronounce the disease to be epilepsy. In a great number of cases of idiopathic epilepsy, the long history of recurring convulsions with no associated symptoms will serve to distinguish it, whereas in *local disease of the brain* there will probably be other indications, such as headache, vomiting, optic neuritis, or local paralyses. In *Bright's disease* the convulsions are epileptiform, but the patients, as a rule, show good evidence of their state of health, in albuminuria, high tension of pulse, hypertrophy of heart, preceding uræmia or œdema; the fits are often ushered in by drowsiness and muscular twitchings, are of much longer duration, and recur frequently in the same day with intervals of drowsiness or semi-coma. Any source of *peripheral irritation* should be inquired into, such as a decayed tooth, intestinal worms, and in children dentition, phimosis, constipation of the bowels, pins in the clothes, &c.

It remains to be mentioned that nocturnal attacks of epilepsy may be for a long time unrecognised, if they are not actually witnessed by any one. They may be suspected if a boy or girl, not suffering from nocturnal enuresis, and beyond the age at which that is usual, unexpectedly wets the bed; or if there are petechiæ on the face or body, or a sore tongue which the patient cannot account for, or headache.

Prognosis.—Epilepsy rarely recovers without treatment, and

the hope, so often entertained by the patient's friends, that attacks beginning in youth will cease with the development of puberty, or with the appearance of the menses, is nearly certain to be disappointed. They can, however, be very markedly controlled by treatment, and are generally the more amenable the later in life they have begun. Gowers states that the prognosis is better if the fits occur only during waking, or only during sleeping hours, and not under both circumstances; if there is no considerable mental change; if the attacks are only of the major kind, and not both major and minor; and that it is better if there is an aura than if there is none.

The effect of the fits upon the mental condition of the patient is, as a rule, directly in proportion to the duration of the illness and the frequency of the attacks.

The fatal terminations of epilepsy have already been mentioned (*see* p. 385); and cuts, bruises, and fractures may be the less serious results of similar accidents.

Treatment.—The object of treatment is to reduce the frequency or prevent the recurrence of the fits. The management of a patient during a fit has also to be considered.

In the Interval.—Something may be done in many cases by careful attention to diet and other hygienic matters. Food should be light and digestible, with a minimum of meat; large meals should be avoided, and especially heavy suppers just before going to bed. Stimulants, including tea and coffee, should be prohibited. Starr recommends the use of intestinal antiseptics (salol, naphthaline, benzoate of soda) if the presence of indican and skatol show the existence of intestinal toxæmia. Regular but not exhausting exercise, and an occupation that does not involve danger to life, if a fit occurs, should, if possible, be obtained; and the treatment of cases in so-called *epileptic colonies*, where the habits, diet, exercise, and recreation are systematically ordered, has been found to present many advantages. Children with epilepsy should be educated suitably to their position in life, but should not be forced into school competition.

Of medicines, the most useful are the bromides of potassium, sodium, and ammonium, which have a very powerful influence upon the course of the fits in the majority of cases. The potassium salt is the one most generally employed, and should be given in doses of from 10 to 30 grains three times a day. For milder cases and young subjects the smaller dose may suffice. Severe cases will require the larger dose, or even more. A combination of the salts is preferred by some, such as the bromides of potassium and sodium, or of sodium and ammonium, or of all three; the dose of the combined salts being the same as that of either given separately. Bromide of strontium is also a useful salt, and is given in similar cases. In any case the remedy must be continued for several months, or years. Its effect is generally to diminish the frequency or the severity of the fits, so that they occur at intervals of months,

instead of every week; if the bromide is left off, the fits again become more frequent, and consequently the patient is glad to continue the use of the drug as a part of the daily routine. Even if the fits cease entirely for several months or a year, the bromide should be continued for two years after the last fit, and then in gradually diminishing doses for another year.

Very large doses of bromide cause dulness and lethargy, with muscular weakness and cold extremities. This condition is known as *bromism*, and is liable to be induced by doses larger than half a drachm three times a day. If it occurs the drug must be diminished in quantity, or stopped altogether for a time; but Dana says it can be very much reduced by continuing the bromide with glycero-phosphate of soda amounting to 20 or 30 grains daily; and nux vomica is also used for the same purpose. The bromide is best taken in plenty of water, and the eruption of acne which sometimes occurs from its use may be prevented by adding three or five minims of liquor arsenicalis.

Several other drugs have been used for epilepsy; either in combination with the bromides, or replacing them from time to time, for instance, when bromism has been induced. They are belladonna, zinc sulphate, oxide, or lactate (up to 10 or 15 grains three times a day), iron, borax (15 to 30 grains), calcium lactate (15 grains), antipyrin, digitalis, and cannabis indica; but none is so good as the bromides.

During the attack.—In cases where there is a definite aura the attack can sometimes be arrested. If the aura consists of a sensation in the hand, which gradually proceeds up the arm, the fit may possibly be checked by vigorously rubbing the part, or by tightly constricting the arm above the seat of the sensation, thus preventing, as it were, its progress to the centres. Where this is successful the patient may wear a cord looped round the upper arm, with one end conducted down his sleeve to the wrist, so that by pulling upon this end he can at once constrict the arm. Other patients ward off fits by lying down on feeling the aura, or by answering the indication afforded by the aura, as in the case of a patient of Strümpell's whose fits were preceded by a sensation of tenesmus, and might be sometimes checked by her going to stool. Dr. Campbell Thomson suggests that in the above instance the attention and mental effort are the causes of the inhibition of the fit, rather than the actual constriction of the arm; and he urges that patients with an aura should be educated to resist the attack by a muscular effort, or by forced attention to surrounding objects. Occasionally the inhalation of nitrite of amyl will prevent the further development of an attack. When the fit has really begun little can be done in the way of treatment; but the patient can be protected from some of the results of the convulsions. As a rule he must lie where he falls, unless this is in itself a position of danger (a pool of water or the fire), but he can be prevented from injuring himself against surrounding objects; his collar, necktie, cuffs, and other tight bands

should be at once loosened; and a piece of cork, gutta-percha, or firewood should be held between the teeth to prevent the tongue being bitten. False teeth worn by an epileptic should always be removed at night, as they may be loosened in a fit and become impacted in the pharynx.

For the *status epilepticus*, the inhalation of nitrite of amyl is strongly recommended by Sir Crichton Browne; other measures are chloroform and ether inhalations, chloral in 15-grain doses every four hours, small morphia injections ($\frac{1}{10}$ grain), a moderate venesection, and ice to the spine.

INFANTILE CONVULSIONS

Convulsions occur with much greater readiness in infancy than in later periods of life, and under somewhat different circumstances. The higher centres are less developed and exert less controlling power over the lower. The circumstances under which convulsions generally occur are the following: (1) The onset of acute diseases, such as scarlatina, measles, and pneumonia; the convulsions here seem to take the place of the rigor of adults. (2) Local diseases of the brain, of which acute meningitis is the most frequent; but tubercular tumours, chronic hydrocephalus, and lesions following otitis are occasional causes. (3) Great exhaustion, as after prolonged diarrhœa, or diarrhœa and vomiting; the resemblance of this condition, formerly called hydrocephaloid disease, to acute meningitis, has been already mentioned (*see* p. 358). (4) Venous congestion of the brain, such as may be caused by an attack of whooping-cough, which sometimes terminates in general convulsions. I have seen a child cry itself into convulsions at once if its mother left it, holding its breath, and becoming more and more livid, until the fit began. Convulsions which not infrequently occur at the end of pneumonia may sometimes belong to this group. (5) Rickets is now held to be responsible for the majority of cases of infantile convulsions not included in the above groups. Often the fit is induced by some peripheral irritation, such as indigestible food; intestinal worms, especially lumbrici; cutaneous irritation, such as pins in the clothing, instanced by Trousseau; or the process of dentition. Perhaps too much has at different times been made of teething as a cause of convulsions, since delayed dentition is a constant result of rickets, and so would co-exist with convulsions, in a large proportion of the cases. Sometimes no exciting cause for the fit can be discovered. (6) Some infantile convulsions must be regarded as really epileptic, since epilepsy may begin in infancy; especially those must be so regarded which commence in early childhood—*e.g.*, at two or three years, when the influence of rickets is beginning to wane.

Of these six groups, it is especially the last two that are usually considered as infantile convulsions proper, or *eclampsia infantum*, the

convulsions in the other cases being more definitely symptomatic. Convulsions in children may closely resemble the epileptic fit of adults; but in a large number of instances they are less complete. They often begin with a short tonic stage: the eyes are turned to one or other side, the pupils are dilated, the head is drawn back, and the arms and legs are rigidly extended. The face may be at first pale, but the lips soon become livid. Twitching then begins in the lips or eyelids, and extends to the whole body, which may be thrown into violent clonic convulsion. The fit lasts a few minutes, and is followed by recovery; or there is a succession of fits, alternating with coma, during which slight twitching of the facial muscles or extremities may take place. Often the convulsion is very much slighter, and consists of little more than deviation of the eyes, or squinting, or fixation of the chest with commencing lividity of the lips, or the convulsive closure of the glottis, known as *laryngismus stridulus* (*see* Diseases of the Larynx); or the hands are extended and rigid, with the thumbs turned into the palms, or the hands and feet are disposed in the manner characteristic of tetany (*see* p. 410). Convulsion may be followed by temporary hemiplegia in children as in adults. Strabismus is another occasional result. Finally, convulsions in children are not infrequently fatal.

Diagnosis.—The recognition of infantile convulsions is not itself difficult. It is necessary, however, to determine upon what they depend. If it is a first fit the possibility that it is the onset of an *exanthem* must be remembered: the temperature should be taken, and the child carefully watched for a time afterwards. Fits due to *cerebral disease* are more likely to be unilateral, and may be accompanied with other symptoms, such as headache, vomiting, retracted abdomen, or optic neuritis. In other cases the indication of rickets must be sought for in the beaded ribs, the enlarged epiphyses, open fontanelle, and delayed dentition; and careful inquiry should be made after some source of irritation, such as unsuitable food, and others above mentioned.

Treatment.—This, as in epilepsy, consists of the treatment of the fits, and the means to be taken to prevent recurrence.

When a fit occurs it is usual to place the child at once in a warm bath. If the bowels have not been recently opened, or if there be reason to suppose the ingesta are causing irritation, a grain or two of calomel may be placed on the tongue. If the fits are very violent and continuous, chloroform may be cautiously administered. It will promptly check the convulsions; but they will probably return soon after it is withdrawn, when it may be again given for a few minutes. When the child recovers sufficiently, 5 grains of bromide of potassium may be given; or if the fits are continuous, it may be given in somewhat larger doses (7 or 10 grains) by the rectum. Chloral may be combined with it to the extent of 3 to 5 grains.

To prevent the recurrence of the fits, one must deal with the predisposing condition, and with the special susceptibility to

convulsion. If an exanthem, meningitis, cerebral disease, or whooping-cough is the cause of the fit, the disease must be dealt with as advised elsewhere, and the fits accompanying such illness are very little amenable to special treatment. The frequency of convulsions in rachitic children can be much influenced by treatment suitable to this disease, such as regulation of the food, administration of cod-liver oil, and general hygienic improvement, together with the use of potassium bromide in doses of 2 or 3 grains three times daily. Similarly, the cases that are more allied to epilepsy should be treated by the regular use of the bromides.

MIGRAINE

(*Megrim, Sick Headache, Hemicrania*)

This complaint consists of recurring attacks of headache, preceded by certain abnormal sensations, and often associated with nausea and sickness.

Ætiology.—It is undoubtedly hereditary, and, like epilepsy, it may have hereditary connections with other neuroses; or it may occur in those who inherit gout. Though it may begin in early childhood, it commonly first appears about the age of puberty, and lasts through the greater part of life; it rarely commences at an advanced age. It is, perhaps, more frequent in women than in men. The attacks are more likely to occur in those whose occupations are sedentary, whose work is chiefly mental, or whose hygienic surroundings are bad. The immediate cause of an attack is often some disturbance of digestion by a large meal, or indigestible food, or by constipation, or it is some exceptional mental or bodily fatigue, or worry or anxiety. Overstrain of the eyes, as in long reading, or in sight-seeing (theatres, picture-galleries), is a not uncommon cause, especially if there is any uncorrected error of refraction; thus, hypermetropia, astigmatism, and muscular asthenopia, which are frequently the cause of frontal headaches, occasionally lead to definite attacks of the special kind of headache known as migraine.

Symptoms.—A complete attack consists of the sensory phenomena and the succeeding headache; but sometimes the attack consists only of headache, and at others the sensations are experienced alone without being followed by pain in the head. The most characteristic commencement of migraine is by a visual sensation consisting of half-blindness or *hemianopia*. In a great number of cases it takes place as follows: the patient may be apparently in perfect health when he notices that he is unable to see what is directly in the centre of the field of vision, but has to shift his head a little for the purpose; or he sees at once that the centre is occupied by a bright spot. In a few minutes the area of blindness enlarges, and if he turns towards a dark part of the room, or to a clear surface, like the ceiling, he will see a small circle of

sparkling colours, having a zigzag or vandyked pattern. From minute to minute the circle grows larger and larger to one or other side and away from the centre, opening out in the form of a horseshoe, which obscures the field of vision as it spreads outwards, while vision returns in the centre and progressively improves. The outer margin of the horseshoe consists of a zigzag line of colours; within this is the blind area, full of movement as of a boiling fluid; within this again, the recovered area of vision. Generally, in half an hour from the beginning the horseshoe has reached the periphery of the field; every object is now visible, though, perhaps, with a sensation of oscillation or quivering. By closing each eye alternately it will be seen that the sensation is not due to an affection of either of them, but to a disturbance of the brain which must be situate behind the optic chiasma on the side opposite to the blind part of the field. This curious spectrum has been described as *teichopsia*, or fortification figures. The visual phenomenon is not always so definite as this—it may consist only of flashes of light or half-blindness without spectra.

Other *sensory* disturbances occasionally occur, and they generally follow the hemianopia in the course of half an hour or an hour, or they may occur without it. They consist of tingling sensations in the limbs, face, tongue, or other parts; they may begin in one finger, spread then to another, and so to the hand or up the arm, and to the face or throat, the part first attacked recovering as the others are invaded. These sensations are mostly unilateral; they may be followed by transient weakness. Aphasia is another disturbance which may occur in the course of migraine; words are forgotten or misplaced, the condition resembling that of one who has just had too much wine. Aphasia is commonly associated with a spectrum on the right side of the field, and if tingling co-exists that also is on the right side; indicating, therefore, a left-sided lesion, as is common in cerebral embolism or hæmorrhage, when speech is involved.

Besides the transient weakness above mentioned, more pronounced *motor* symptoms have been rarely observed, in the form of a temporary monoplegia, hemiplegia, or motor aphasia. This has occurred in several members of a family (J. M. Clarke).

In some cases the only disturbance preceding the headache is of a *mental* kind; there is depression, languor, or fear of impending evil; and these feelings may last from half an hour to two or three hours.

The more distressing feature of the illness is the headache which comes on sooner or later after the preceding sensations, generally as these are declining; sometimes even the day after the spectrum, with a perfectly healthy interval. The headache is of all degrees of intensity; so slight as not to interfere with even mental work, or so severe as entirely to unfit the sufferer for any action whatever. It often lasts a whole day, and in the severer forms it increases gradually, until when at its height nausea and vomiting occur, and a certain amount of relief is afforded to the pain. After this it

quickly disappears, or it subsides more gradually, or it continues till the patient seeks his bed at night; and he awakes, perhaps after a prolonged sleep, cured, but with some sense of weakness or fatigue. The pain is often unilateral (*hemicrania*), but it may begin on one side and change over to the other, and even return again to the side first affected, or it may affect both sides at once; the side first affected is generally that which is opposite to the visual spectrum. The pain may begin in the frontal, temporal, or parietal region, or behind the eye, often at a very limited spot; whence it may spread in different directions, or become general. It is often boring in character, and aggravated by movement, light, or sound; and the patients are only comfortable in the recumbent posture. The pain may be so bad that the patient passes into a stupor or becomes delirious. With this the face is pale and drawn, the hands and feet cold, the pulse feeble, small, and slow. Only in some cases it appears that as the headache continues these conditions are reversed, the face flushes, and sweating occurs.

The attacks of migraine recur at intervals of a few days or two or three months, an interval of three or four weeks being more common. Particular attacks may be determined by the special causes enumerated, but sometimes it is impossible to find out what has induced the disturbance. The disease often lasts throughout a long life, although it sometimes becomes less frequent, or disappears altogether, after the age of fifty. Cases have been observed in which attacks of migraine have become less or ceased on the appearance of other neuroses, such as epilepsy, asthma, or spasmodic croup; and a similar relation to gout has been also shown to exist.

Pathology.—The popular idea that this is a gastric or "bilious" disorder is perpetuated by the use of the term sick headache, by the vomiting of bile which sometimes occurs, and by an attack being occasionally induced by an injudicious meal. But the latter cases form a very small proportion of the whole, and it is clear, from the preceding visual and other sensory phenomena, that it is quite early or primarily a lesion of the cerebral cortex, involving, it may be, a large extent (centres for vision, speech, sensation, and motion). Several authorities have advocated a vasomotor theory of its origin; for instance, vascular contraction might cause anæmia, and so hemianopia, and subsequent dilatation might account for headache by engorgement; but it remains an unproven hypothesis. Gowers points out that the disturbance is double; the blindness is due to inhibition, the spectrum to discharge. In any case it is certain that no coarse anatomical change is present as a cause of the disease.

Diagnosis.—The association of the headache with the various sensory disturbances, and its recurrence at intervals, are generally sufficient to distinguish the complaint. The headache of brain-disease is either continuous, or, if it remits, the intervals are shorter, and the attacks longer than those of migraine. Where the visual spectrum is present, it is generally quite characteristic. Epilepsy

with a visual aura may be confounded with it; but the aura of epilepsy is of very short duration, while the spectrum of migraine mostly lasts from twenty to thirty minutes. There is no loss of consciousness in migraine.

Prognosis.—Under treatment much improvement may be obtained both in the frequency and in the severity of the attacks, though the disease commonly continues for years. It is, however, not dangerous to life, and there is no evidence that sufferers from migraine are more liable than others to hæmorrhage, thrombosis, and other diseases of the brain.

Treatment. *In the interval.*—The patient should be placed under the most favourable hygienic conditions, including a carefully regulated diet, the avoidance of constipation, exercise without exhaustion, pure and bracing air, and exemption from excessive brain study or mental worry. To these may be generally added the use of tonic remedies, such as iron, quinine, strychnia, arsenic, and cod-liver oil. Ocular defects, if any, should be remedied by suitable spectacles. Seguin, of New York, strongly advocated the use of cannabis indica to prevent the recurrence of attacks; it is best given twice a day in pills containing $\frac{1}{3}$ or $\frac{1}{2}$ grain of the extract, and these should be continued, like any other treatment employed with this object, for at least six months, and, if necessary, for longer. Bromide of potassium is uncertain in its action: it is most successful, according to Gowers, in cases in which the face flushes or is unchanged in colour. For cases in which pallor occurs he recommends nitro-glycerine to be taken two or three times daily after food, in doses of $\frac{1}{200}$ to $\frac{1}{150}$ of a minim; he suggests that by frequently flushing the brain with blood the nutrition of the parts concerned may be improved.

During the attack.—If the headache is severe the patient should lie down in a darkened and quiet room, with a cold-water compress to the head, and hot-water bottles to the feet if there is a tendency to collapse. He may take soda water, or suck ice; there will be little desire for food, but after a time some soup or beef-tea may be beneficial. Antipyrin is a valuable remedy in sick headache, and may be given in doses of 5 to 15 grains. Phenacetin (3 to 10 grains) has a somewhat similar action. Many people find relief in tea, or coffee, or guarana, a substance which contains caffein in greater proportion than either tea or coffee. It may be taken in three or four doses of 15 to 30 grains mixed with water, at intervals of half an hour.

Failing these, bromide of potassium (20–30 grains), chloral hydrate (15–30 grains), butyl-chloral hydrate (10–15 grains), or cannabis indica, in doses of 10 minims of the tincture, or $\frac{1}{2}$ grain of the extract, may be tried: of these, the first is most likely to do good. Nitrite of amyl and nitro-glycerine on the one hand, and ergotin on the other, have been given to influence the vasomotor system, and have occasionally done some good, but they cannot be depended upon. Locally, besides cold applications various anodynes

have been employed with varying success, such as ether or bisulphide of carbon on cotton wool covered by a watch-glass, extract of belladonna, diluted ointment of veratria, and menthol.

VERTIGO

This is a disorder of the function of equilibrium of the body, by which is produced a sense of unsteadiness or of movement of the body in one or other direction, or a sense of movement in surrounding objects, or an actual movement of the body itself. The term is more or less synonymous with *giddiness* or *dizziness*.

It is of very different degrees. It may amount only to a slight instability or unsteadiness. Sometimes the patient seems to fall forward, or turn round, when he actually does not move (*subjective vertigo*); in other cases, surrounding objects appear to be moving round, or up and down (*objective vertigo*); or the sense of movement of the body and of surrounding objects may occur together. Often the patient reels and staggers, takes hold of a chair, table, or railing for support, or, failing that, may fall to the ground.

The equilibrium of the body depends upon a reflex system of which the middle lobe of the cerebellum forms part. The centripetal impressions are derived from the skin, from the muscles, especially of the lower part of the body, and from the semicircular canals of the labyrinth of the ear (*see* p. 325). A knowledge of the position of the head and eyes also contributes to equilibrium, and this is probably derived from active innervation. If any one of these centripetal impressions is deficient, vertigo may result, and one of the most common causes of vertigo is a lesion of the labyrinth, producing what is known as *labyrinthine* or *aural vertigo*. Experiments on animals have shown that lesions of the semicircular canals will produce vertigo, the direction of the movement being determined by the canal injured—whether horizontal, transverse, or vertical, whether right or left; and by the nature of the injury —whether irritative or destructive. But vertigo in man is caused not only by primary disease of the labyrinth, such as hæmorrhage or congestion, but by diseases of the middle ear, and of the meatus, such as inflammation of the tympanum, retracted tympanic membrane, obstruction of the Eustachian tube, and collections of cerumen in the meatus; and even by syringing the ear, when the membrana tympani is perforated. Probably the effect is brought about by these morbid conditions altering the pressure of the endolymph in the labyrinth.

It is, however, the internal ear which is affected in the great majority of cases; the patients suffer, at the same time, from tinnitus, deafness, and vertigo; and the deafness is not due to imperfect conduction through the middle or external ear. It was in some very severe forms of aural vertigo that Menière called attention to the association of the symptoms, and the presence of

disease in the semicircular canals; hence the term *Menière's disease* has been employed, and has been extended by most writers to every form, however slight, of labyrinthine vertigo. In well-marked cases there is a constant tinnitus, with more or less deafness, of which, however, the patient may himself be unconscious, until it is shown by special examination. The vertigo is generally paroxysmal, occurring at intervals of some days or weeks, excited by movements, and by coughing, sneezing, or blowing the nose; or it occurs spontaneously, or even during sleep. The attack sometimes begins with great increase of the tinnitus, which may resemble the whistle of a locomotive, the firing of a gun, or the roar of a waterfall. The vertigo may be of any of the kinds above mentioned; the patient may appear to turn round, or be thrown forwards, or he may see objects moving round, or to one or other side, or he may himself fall forwards on to one side. The movement, or the sense of movement, both of the patient and the surrounding objects—for these, as a rule, coincide in direction—is generally towards the side of the affected ear. There may be a very short interval of loss of sight or of unconsciousness, and, in bad attacks, there is nausea, followed by vomiting, pallor, and coldness of the extremities. Sometimes there are movements of the eyes in aural vertigo. The sense of giddiness may last for two or three or several hours, so that the patient is totally unfit for anything, and has to confine himself to the recumbent position; and tinnitus and deafness persist for some time also in an aggravated form.

The attacks in progressive cases may become almost continuous, so that the patient is always suffering more or less from vertigo, and is unable to move at all, while, from time to time, fresh paroxysms increase his sufferings. But if deafness becomes complete the vertigo generally ceases.

Ætiology.—Aural vertigo is rare under thirty years of age: the conditions which lead to it seem to be cold, gout, syphilis, arterio-sclerotic and senile changes. The attacks are excited not only by movement, but also by fatigue, exhaustion, and gastric derangements.

Pathology.—The exact pathology of labyrinthine vertigo still remains uncertain. By some it is thought that the lesion may be central, occurring in the auditory centres or in the medulla oblongata, and that the symptoms are referred to the ear, as the ocular symptoms of migraine are referred to the eye. On the other hand, it appears certain that the labyrinth is sometimes the seat either of primary disease or of such secondary disturbances of pressure as may result from inflammation of the tympanic cavity. But probably, when the lesion is confined to the middle ear, the case should not be termed Menière's disease, and still less if it is in the external ear. The paroxysmal occurrence of the attack in the course of persistent chronic disease has yet to be accounted for.

Vertigo also occurs in some cases of epilepsy, and in migraine; it

may result from organic disease of the brain, such as tumour; or from *defective muscular sense* in locomotor ataxia. Rarely an *ocular vertigo* results from weakness of one of the muscles of the eyeball; the patient forms a wrong impression as to his surroundings, and the sense of equilibrium is disturbed. *Laryngeal vertigo* (Charcot) is another rare form. The attack begins with burning and crushing pain in the larynx, followed by sharp dry cough. During the cough vertigo occurs, and the patient may become unconscious, and even have convulsions. A true *gastric vertigo* is much less common than aural vertigo, and has been attributed both to reflex irritation of auditory fibres, and to absorption of toxins: but in many cases supposed to be gastric, the permanent cause lies in the aural apparatus. Vertigo also arises from *anæmia* of the brain, from the action of some *drugs*, and from *auto-intoxication* (gastric, uræmia); and from *psychical* causes, *e.g.*, the giddiness caused by looking down from a height. Allied to this is the vertigo which may occur both in *neurotic* and *neurasthenic* subjects, of which fatigue, overwork, or excitement may be the immediate cause. *Senile vertigo* is often a mild form, common in elderly people, and probably related to arterio-sclerotic changes. Cases attributed to gout and to syphilis may perhaps be explained by similar arterial changes.

Treatment.—Potassium bromide is of great value, and should be given in doses of 15, 20, or 30 grains three times a day; it probably acts by lessening the conditions of instability of the centre. It may be combined with potassium iodide and hydrobromic acid. Quinine has been given in doses of 7 to 15 grains daily for some months with success. Sodium salicylate in 5-grain doses thrice daily, aspirin and phenacetin are also useful. Blisters or stimulating ointments may be applied behind the ear. Where syphilis or gout is likely to be the cause, it should be met by appropriate remedies, and when the vertigo is associated with conditions of high arterial tension, this should be treated by aperients, and especially by mercury, in the form of blue pill or calomel. The vertigo of Menière's disease has been cured in some cases by the removal of the labyrinth.

In other forms than aural vertigo the removal of such causes as can be found, and the use of bromides, are the chief indications. Ocular vertigo is prevented by closing the affected eye.

PARALYSIS AGITANS

(*Shaking Palsy. Parkinson's Disease*)

This disease consists, in its fully developed form, of rhythmical contractions of the muscles of the limbs, associated with weakness and rigidity.

Ætiology.—It is a disease of advanced life, rarely occurring before the age of forty-five years; but, on the other hand, not often

commencing after the age of sixty-five. It occurs in men twice as often as in women. It cannot always be traced to any definite cause; emotion, fright, injuries, acute diseases, and exposure to cold have been the determining factors in some cases.

Symptoms.—It commonly begins in one hand and arm with a tremulous movement, due to rhythmical contractions of antagonistic muscles. The movements are most marked in the hand; the fingers are generally flexed, with the thumb resting against the forefinger, and the constant slight flexion and extension of the fingers and thumb produces a movement like that required for rolling pills. Similar slight movements of flexion and extension occur at the wrist and elbow joints. After the tremor has existed for some time in one arm, it generally spreads to the leg of the same side; and then in succession to the arm and leg of the opposite side. The trunk may also be affected, though it is not always easy to say how much the tremor is due to the movements in the legs; and, finally, in some cases, there is a slight movement of the head. Occasionally even the muscles of the jaw and tongue, but very rarely those of the face, are affected. These movements vary in extent; in rapidity they range between $4\frac{1}{2}$ and 7 to the second. As a rule the movements continue even during rest; thus, if the patient sits with the arm resting on the knee, both the leg and the hand and arm will continue to tremble. In early cases, however, support may check the tremor for a time, and in advanced cases with the rigidity to be presently described, the tremor may only occur on movement. By voluntary efforts fixing the limb, the tremor may also for a time be stopped, and it ceases during sleep.

Another feature of the disease is weakness and rigidity of the affected muscles. In the majority of cases this follows the tremor: in others, it occurs at the same time as, or before, the tremor. In a case which has begun with tremor in one limb, the rigidity may appear first in another limb, and be followed by trembling. The muscular weakness is shown by deficient power of grasp, and fatigue on exertion. The rigidity imposes on the patient a characteristic posture, which is most marked when he is standing. The body is bent somewhat forwards, the elbows are flexed nearly to a right angle, and stand out a little from the side, the hands are in the position above described; the legs are slightly bent at the knees; the head is also inclined forwards, and the face wears a fixed, anxious expression. The gait is very peculiar; the patient rises from his seat slowly and with apparent difficulty, and his first steps are hesitating; but soon his movements become quicker and quicker, he seems with each step to be trying to prevent a fall, and ultimately, unless stopped, he may actually fall forward to the ground. Some patients when gently pushed backwards are unable to stop themselves, and continue to walk backwards until they meet an obstacle or fall. The terms *festination* and *propulsion* have been used to describe the forward tendency; *retropulsion* and *retrogression*, the backward movement. It has been noticed as an early

symptom that the toes are curled under the foot when the patient begins to walk (P. Stewart). Speech may be similarly slow at first, and afterwards rapid; it is often high-pitched or thick and feeble. Otherwise all movements tend to be slow on account of the rigidity: for instance, the patients turn with difficulty. The muscles are not hypertrophied by their excessive action, and only in late cases with long-continued rigidity do they present some atrophy. The reflexes and electrical reactions are generally normal. Some subjective sensations are often experienced, such as dull, aching pains, sense of fatigue, or a feeling of restlessness, and especially a sense of great heat, which is often accompanied by free perspiration; while a flush on the cheek of the patient shows vasomotor weakness.

The disease is chronic and progressive, yet its course may be very slow, two or three years perhaps elapsing between the affection of one and another limb. Probably a well-marked case never recovers; but it is fatal only through bed-sores or exhaustion in the extreme cases; intercurrent disease—*e.g.*, of the lungs—terminates others.

Pathology.—Neither the situation (whether cerebral, or spinal) nor the nature (whether structural, functional, or auto-toxic) of the lesion in this disease is as yet known. Sclerotic patches in connection with vessels in the spinal cord have been described, and the anterior cornual cells are atrophied and pigmented to a greater extent, according to Dana, than senility would account for. He suggests lesions in the anterior cornual synapses, or possibly in the mid-brain (*see* p. 324).

Diagnosis.—There is but little difficulty in recognising paralysis agitans. Formerly, no doubt, it was confounded with *disseminated sclerosis*, which occurs, however, earlier in life, and in which there are wider and more irregular movements of the limbs brought on by movement only, implication of the head, staccato or scanning speech, and nystagmus. *Senile tremor* is distinguished from paralysis agitans by the constant trembling of the head. The movements also are finer, both arms are often involved together, and there is no rigidity: it comes on later in life.

Treatment.—This is most unsatisfactory. Business worry and excitement should, of course, be avoided. Various sedative drugs—morphia, codeia, opium, hyoscyamine hydrobromide, hyoscine (scopolamine) hydrobromide ($\frac{1}{200}$ gr. to $\frac{1}{50}$ gr. subcutaneously), and duboisin sulphate ($\frac{1}{100}$ gr.)—may check the movements for a while, but without permanent benefit. The last two are the most useful. Arsenic seems to have done good in some cases, and Gowers recommends arsenic, Indian hemp, and opium together. The continuous galvanic current, Swedish movements, and massage have also benefited some cases.

CHOREA

(*Sydenham's Chorea. Chorea Minor*)

Chorea (χορεία, a dancing) is characterised by irregular involuntary movements of different parts of the body. The popular equivalent, St. Vitus's Dance, has reference to the occurrence in the Middle Ages of epidemics of dancing mania, when patients were cured by a pilgrimage to the shrine of St. Vitus—Chorea Sancti Viti. But the complaint in those epidemics partook rather of the nature of hysteria, and though the name chorea is still sometimes used to indicate some other forms of abnormal movement (*chorea major*), it is, as a rule, reserved for the disorder now to be described.

Ætiology.—It is mostly a disease of childhood : nearly half the cases occur between the ages of five and ten, and another third between ten and fifteen. It is more frequent in girls than in boys, in the proportion of two or three to one, and it is more common among the poorer classes of society. It is not strongly hereditary in its ordinary form. Among antecedent diseases, acute rheumatism is the most important. About one-third of choreics have had rheumatic fever; choreic movements sometimes occur in the course of rheumatism, or rheumatic pains during chorea. In some other cases of chorea the attack has been preceded by one of the infectious disorders, such as scarlet fever or measles; or some other septic disorder. Among adult patients, pregnancy is a common antecedent: some of them have had rheumatism, and others chorea in childhood. Fright or mental shock of some kind is certainly a cause of the disease in many cases, though parents are often too ready to account for the attack in this way. It may arise after injury, perhaps also as a result of emotion. The origin of chorea in imitation is probably a very rare event.

Symptoms.—The most prominent feature of the disease is the action of the muscles: they are in a condition of (1) involuntary movement, (2) ataxy or inco-ordination, and (3) slight degree of actual weakness or paresis. The patient is in a constant state of movement, whether lying, sitting, or standing; and the movements, which affect nearly all the muscles of the body, are jerky, irregular, and devoid of purpose. The fingers are opened and shut, the wrist suddenly extended or flexed, or the shoulder lifted. The facial muscles are twitched, the eyebrows suddenly elevated, the head or the eyes rotated to one side, and the chin elevated or depressed. In the lower extremities the movements are often less; the toes are twitched, or one knee gives way. In the muscles of the trunk, one notices half rotation of the body to one or other side, sudden retraction of the abdomen, or jerky action of the respiratory muscles.

The irregularity is more marked on voluntary movements. If the hands are stretched out in front, the child is quite unable to

hold them steady; on protruding the tongue, it is put out with a jerk, and perhaps withdrawn suddenly, and the muscles of the jaws act capriciously at the same time; in walking the legs are thrown about, the body is jerked round, and the shoulders are lifted. In the same way it may be seen that the muscles relax with great readiness; after grasping an object, one or two fingers quickly yield, and soon the hand and arm will drop. The movements are increased when the patient is watched, or if she becomes excited; they cease during sleep.

The vocal cords have been seen to quiver, and a low-pitched, monotonous voice is attributed to their want of tension. Speech is irregular, and the patient is unable to sing a long note; these may be due to the irregularity of the respiratory movements.

Sensation is but little disturbed; there may be some formication or tingling, but very rarely any definite hyperæsthesia, or anæsthesia. Both nerves and muscles show increased irritability to faradic and galvanic currents.

It is not always easy to say what the condition of the mind is: often a child with chorea looks silly or idiotic from the purposeless contractions of the facial muscles, which in this case are not a true index of the mind. Apart from this, however, the child's disposition is apt to be altered; she becomes fretful, irritable, capricious, or excited, while intellectually she has a weak memory and is unable to fix the attention.

In about half the cases a murmur over the heart's area may be recognised. The heart is also often irregular in action, but this is probably secondary to the irregularity of the respiratory movements. The murmur is commonly heard at the apex of the heart, and is systolic in time. Mostly it is limited to this area; occasionally it is audible in the axilla and behind, and is obviously due to mitral regurgitation. Sometimes a hæmic basic murmur is present. The origin of the apex murmur has been much discussed, but since endocarditis has been frequently found in fatal cases, and some of the murmurs of chorea are undoubtedly due to a valve lesion, it is fair to suppose that in other cases they arise from endocarditis, or possibly from myocarditis. Some, indeed, may be the result of preceding rheumatism; but this will not account for the majority, which appear to develop in the course of the chorea itself.

Varieties.—Sometimes the symptoms are very slight, and remain so for some time; the fingers are only twitched a little, irregular movements are scarcely noticed, but the child drops things that she attempts to carry. In some cases the movements are limited to the arm and leg of one side only (*hemichorea*).

In others there is decided paralysis, with only slight choreic movements; the arm hangs by the side, and can with difficulty be raised; the fingers are twitched occasionally, and the grasp is extremely feeble (*paralytic chorea*).

Exceptionally the movements are very violent; standing or sitting is impossible, and the patient is confined to bed, where she throws

herself about in the wildest contortions, striking the hands and arms against the sides or head of the bed, and rubbing the elbows, shoulders, buttocks, hips, knees and heels, so as to produce serious abrasions of the skin. Feeding becomes difficult or impossible, as everything placed to the mouth of the patient is jerked aside or spilt; and even if it gets into the mouth it may be rejected by the want of co-ordination for deglutition. These cases (*chorea gravis*) sometimes progress with great rapidity; the patient appears to be exhausted by the constant movement and the want of sufficient nutriment; rapid emaciation takes place, the face is flushed, the eyes sunken but bright, the lips and tongue dry, the pulse rapid, and ultimately death may occur, being preceded often by some rise of temperature and by cessation of the movements. In some the mind is severely affected, and the patient becomes delirious, or even wildly maniacal. Such violent cases are much more frequent in adults between the ages of fifteen and twenty-five, and a large proportion are in pregnant females.

Duration.—The duration of chorea is very variable. The majority of cases last from six weeks to three months; not infrequently slight twitching may occur for many weeks or months after the severer manifestations have subsided, and the symptoms may again after a time become aggravated. In the end most cases recover. The violent cases are usually of short duration; if death takes place, it is often within two or three weeks from the first symptom, or from the time when the movements became violent; if recovery ensues, the movements become quieter after a few weeks, though complete cure may be delayed some time. Chorea is very apt to recur even after its entire subsidence; second and third attacks are frequent. These may be of shorter duration than the primary attack, but are not different in other respects.

Sequelæ.—The disease sometimes leaves behind it a liability to sudden starts, which in the course of months subside. In some cases towards the end of the attack paralysis of the limbs occurs. This may be only on one side (*choreic hemiplegia*); but rarely all four limbs are affected, the child lying quite helpless, and each limb dropping like a log on being raised from the bed. Speechlessness, mental weakness, maniacal and melancholic conditions also occasionally occur, and are generally temporary. Epilepsy has also been observed as a sequel of chorea. The endocarditis may terminate in chronic valvular disease.

Morbid Anatomy.—The nervous system after death does not present to the naked eye any morbid appearances. The microscopic changes which have been found vary in different cases, and are probably either accidental or secondary. Among these may be noted obstructions of minute vessels in the brain, swelling and degeneration of nerve-cells in the corpora striata and other parts, enlargements of the perivascular spaces, and hæmorrhage around minute vessels. Of the other organs of the body the heart is the only one that is generally involved; and in fatal cases of chorea this nearly always

presents evidence of endocarditis (75 out of 80 cases, Sturges; 17 out of 18 cases, Fagge). Fine granulations are found along the edge of the mitral valve on the auricular face, and sometimes on the aortic valves. These are present even when there has been no antecedent rheumatism. Occasionally the valvular lesion is older and more extensive.

Pathology.—The seat of the lesion and its nature have to be considered. Its origin in the brain, and indeed largely in the motor convolutions, is shown by its frequent connection with emotional disturbance, the influence of the will, of emotion, and of distraction of the attention upon the movements, their cessation during sleep, their frequent limitation to one side in the limbs, while they affect both sides of the face and trunk, and the coincident disturbance of the mental faculties. It is highly probable that chorea has a toxic or microbic origin. The facts in favour of this are: The frequent occurrence of endocarditis, and its almost universal presence in the fatal cases; the association with rheumatic fever, and possibly other diseases of infectious nature; and the mode of death, which is by no means always explained by simple muscular exhaustion, for the patient may lie for some hours before death perfectly tranquil, and give the impression that convalescence has begun. Chorea would be thus analogous to tetanus and hydrophobia, in which muscular movements are determined by the circulation of poisons, without the presence of demonstrable nerve-lesions to account for the symptoms. Choreic movements have been produced in rabbits by the injection into the veins of Poynton and Paine's rheumatic diplococcus. Other observers have isolated pyogenic organisms from choreic cases.

Diagnosis.—This rarely presents any difficulty. Movements closely resembling those of chorea may occur as a part of *hysteria;* they are generally more rhythmical, more localised, and may recover quickly. *Habit spasm* occurs in children, and is closely allied to the above; the movements are localised, voluntary in character, more under control and less constant than those of chorea. There are jerky movements in *Friedreich's ataxia;* but the gait is different, the history is a very long one, and nystagmus is present.

Prognosis.—In children it is favourable, apart from the condition of the heart; in young adults it is much more uncertain.

Treatment.—The child should be kept quiet in bed, and everything tending to worry or annoy should be kept from her. She should not be subject to the ridicule of companions, nor to much study of lessons. The diet should be plain, nutritious, and abundant. Arsenic is the drug which is most widely employed. It undoubtedly shortens the attack; it may be given in 2 or 3 minim doses of liquor arsenicalis three times a day after meals, gradually increased to 5 or 7 minims in young children, or to 10 minims in those who are approaching puberty. The salts of iron

are also sometimes given. Antipyrin in doses of 5 to 7 grains, and aspirin in the same doses, appear to be of use sometimes. Chloretone in 5-grain doses three times a day has been found to check the movements and shorten the attack. In severe cases in children massage is of benefit in reducing the disease to more moderate limits. In the very violent cases treatment becomes difficult. The patient must be protected from injury by padded boards at the side of the bed, the nutrition must be maintained, and food may have to be given per rectum. Chloral is probably the best drug to administer, but must be given with caution; morphia is less desirable. Trional in 10 or 16 grain doses has been used with success in both these and the milder cases. Quiet may be obtained for a time by inhalations of chloroform, but the movements return as the effect passes off. In cases of paralysis after chorea I have seen good results from strychnia. When the movements are slight in mild cases, or in recovering stages, gymnastic exercises or the skipping-rope may be found useful.

CHOREA IN ADULTS

A disorder somewhat different from the chorea already described occasionally affects persons of middle and advanced age. The movements are generally more extensive, often prevent the individual from following any occupation, and may persist for years; though some cases are of short duration and recover. These cases seem to have no connection with rheumatism or cardiac disease, but may arise from fright or severe emotion.

Under the name of *Huntington's Chorea* is described a form which occurs in several members of the same family, affecting males more than females: it appears late in life, and is associated with definite insanity, which is usually in the form of progressive dementia. Meningo-encephalitis and changes in the large pyramidal cells of the cerebral cortex, together with small-cell infiltration, have been described in some cases.

Two groups of cases have been described as *electrical chorea.* One of these, described by Dubini, begins with pains in the head, neck, and spine, and then occur quick, sharp, muscular contractions in the muscles of one arm, one side of the face, and then of the leg of the same side: finally, of muscles of the other side of the body. Epileptiform attacks and paralysis may follow and coma and death after weeks or months. It occurs at all ages, and is probably to be classed with myoclonus. The other variety of electrical chorea, described by Bergeron, occurs in children, generally recovers, and is probably of hysterical nature.

MYOCLONUS

(*Myoclonia*)

Under this title are grouped a large number of conditions, of which the essential feature is short, quick contraction of muscles, not forming part of epilepsy, hysteria, chorea, athetosis, &c. Dana, indeed, includes chorea (which he calls infectious myoclonia), spasmodic tics, and fibrillary twitchings of all kinds.

PARAMYOCLONUS MULTIPLEX

Friedreich described under this name the case of a man who had sudden lightning-like contractions of the large muscles of the arms, forearms, and thighs. The contractions ceased when he walked, and were worse when he was quiet in bed : if one arm was used the contractions ceased in it, but continued in the other. The contractions, though violent, produced no movement of the limb as a whole. The knee-jerks were increased, the skin-sensibility, muscle-sense, and vasomotor and secretory phenomena were normal. The condition was cured by galvanism, but it relapsed and persisted till the man's death. But numerous other cases have been recorded of clonic muscular contractions which differ in many points from Friedreich's case. The essential seems to be the occurrence of spontaneous rapid contractions of isolated muscles or parts of muscles in different parts of the body ; in some cases the limbs, in others the face and trunk ; generally bilateral, but not of necessity equally, or, at the same time, on the two sides ; sometimes, but not generally, causing locomotive effect in the parts involved ; occurring at the rate of 60 to 100 in the minute, but quite irregularly ; varying in frequency and force on different days ; and often increased when the patient is under observation, or exposed to external stimuli (sound, touch). The mechanical excitability of the muscles is increased, their electrical excitability is unchanged. Some cases have been associated with epilepsy (*myoclonus epilepsy*), others have shown close resemblances to hysteria, and it has occurred in two or more members of the same family ; rarely these movements have been associated with definite disease of the central nervous system. The pathology is unknown. Galvanism to the spine, hydropathic treatment, arsenic and chloral have been used to combat it, and recoveries have occurred ; but the prognosis is not good.

GENERAL CONVULSIVE TIC

A form of clonic muscular contraction possibly allied to the above, but probably much more dependent upon the mental state of the individual, is this complaint, described as *Maladie des tics convulsifs* by Guinon and Gilles de la Tourette. It is characterised by contractions of the facial muscles, by systematic movements of

different parts of the body which always repeat themselves in the same way, by the utterance of strange noises, by repetition of words heard (*echolalia*), by frequent utterance of obscene words and expressions (*coprolalia*), and by imitation of other movements (*echokinesis*). It often begins in childhood, the facial clonus occurs first, and the extension to other parts of the body, and to other kinds of movement, takes place from time to time in the course of subsequent years. It is a very obstinate disease, but improvement has been obtained by the help of bromides, chloral, hydropathic treatment, isolation, and gymnastics.

HABIT SPASMS

This name is given to movements, occurring in children, between five and twelve years of age, affecting especially the facial muscles, and frequently repeated at irregular intervals. Such movements are blinking the eyes, twitching of the angle of the nose, or mouth, shrugging the shoulders, twitching the fingers, uttering noises, or words, kicking out the legs, or other simple movements. They have probably been voluntary in the first instance, for the purpose of relieving an irritation, or in response to some local sensation, and then have been repeated reflexly, or stimulated by a thought and finally have become automatic—a bad habit; of which indeed the child may be almost unconscious. The movements may be less under observation, and in the same child one kind of movement may be cured, and after a time another will take its place. The children are often neurotic, or of feeble health, and the trouble is often started by some local irritation, or emotional disturbance. It may last months or years, but eventually recovers in most cases. The severest cases are probably indistinguishable from the above described convulsive tic. But usually the distinction has to be made from chorea, of which the movements are not localised to one small group of muscles, and are increased by observation. Localised movements in hysteria are kept up under observation, and are more rhythmical, as a rule.

In the treatment of habit spasms, every possible cause of local irritation should be removed, the child should generally be kept from school, and should have country or seaside air. He may be encouraged not to give way to the movements, but must not be scolded or punished. Tonic remedies may sometimes assist.

SPASMODIC TORTICOLLIS

Besides the temporary affection known as stiff neck or rheumatic torticollis (*see* p. 431), there are two more lasting conditions known by the name of torticollis; *fixed torticollis* or congenital wryneck, and *spasmodic torticollis*. The former is due to a permanent shortening of the sterno-mastoid muscle, which is attributed in some cases

to injuries during birth, is observed first during childhood, if not in early infancy, and causes asymmetry in the bones of the face.

Spasmodic torticollis, or spasmodic wryneck, is a functional disorder characterised by tonic and clonic contractions of the muscles of the neck, whereby the head is forced into an abnormal position.

Ætiology.—The disease is rarely seen before the age of thirty; it affects both sexes, but females more often than men. The cause cannot always be ascertained; neurotic inheritance, exposure to cold, falls and injuries, and overstrain of the shoulder, arm, or neck in particular occupations, have been recorded as antecedents.

Symptoms.—It begins gradually, being first felt as a mere discomfort in the neck; then distinct jerking movements of the affected muscles are felt, by which the head is rotated or displaced. If, for instance, the right sterno-mastoid is affected, the head is constantly being jerked in the direction of the action of this muscle, the chin is thrust forwards or upwards to the opposite side, and the occiput is drawn down towards the clavicle. The contractions are sudden, irregular, and frequent; for a few moments there is a remission, during which the patient slowly and cautiously tries to bring the head straight, when the muscle again contracts, and the face is gradually forced round to the left. The movements are for a time checked by supporting the head, and they cease during sleep, but immediately the patient awakes the movements recommence, and continue with but little rest throughout the day; they are generally increased when attention is directed to them. The muscle most frequently affected is the sterno-mastoid, and next to that the upper part of the trapezius and the splenius capitis; the complexus and trachelo-mastoid, the deep rotators of the head, and the platysma myoides are also sometimes concerned. The position of the head is of course determined by the muscles which contract. The sterno-mastoid produces the results already described; the trapezius draws the head backwards and downwards towards its own side, with slight rotation of the face towards the opposite side; while the splenius draws the head downwards with slight rotation towards its own side. Two or more of these muscles may be affected at the same time; most often the sterno-mastoid with the upper part of the trapezius of the same side, or the splenius of the opposite side. An intermediate position of the head will of course be the result. Or corresponding muscles on the two sides may act together, drawing the head backwards during their contraction. In such cases there is generally an associated contraction of the frontal muscles which normally contract when we throw back the head to look upwards. Occasionally the disease itself spreads to the muscles of the face, or to those of the shoulder or arm, especially at the height of the paroxysm, and in rare cases the lumbar or spinal muscles may be affected so that the body is drawn down to one side by the frequent contractions. In slighter cases, or in early stages, there may be no pain, but in severer forms there is neuralgic pain in the contracting muscles. From their excessive action the muscles often

hypertrophy; they at least retain their natural bulk. The electrical irritability is normal, or increased.

Pathology.—Of this little is known. The disease is not due to lesion of muscle or of nerve; but it is a disturbance of the motor centres, either in the cortex of the brain or in the spinal axis, or perhaps in both situations in the same case.

Diagnosis.—The constant movements distinguish this disease from congenital wryneck, with its early history and facial asymmetry; from the temporary disorder, "stiff neck;" and from spasm of the muscles in caries of the cervical vertebræ. Spasmodic wryneck may be simulated by hysteria, but hysteria has often been unjustly assumed when the patient is a real sufferer.

Prognosis is unfavourable; the spasms may subside after a few months, but far more often persist in a more or less severe form for the rest of life.

Treatment.—The disease is most intractable, and numbers of drugs have been tried with but a small amount of success. I have seen good from bromide of potassium. Gowers speaks of benefit from asafœtida and valerianate of zinc. Opiates, chloroform and Indian hemp, may check the spasms while the patient is under their influence, but it returns again in the course of some hours unless the dose is repeated. Galvanism and massage are sometimes useful. For the former a weak constant current should be passed continuously through the contracted muscles for five or ten minutes daily.

In severe or obstinate cases the nerves or the muscles may be divided. If the former are selected, the deep cervical nerves must be resected as well as the spinal accessory. The movements will cease, but some incapacity must result from the accompanying paralysis and atrophy: and the disease may reappear in adjacent muscles. Kocher, of Berne, claims success for his method of dividing in two, three, or four operations, the sterno-mastoid, trapezius, splenius capitis, complexus, trachelo-mastoid, and even the obliquus inferior. Relief has been obtained by the use of a light steel spring clamp to the back and sides of the neck (Hall).

TETANY

In this complaint there are peculiar contractions of the muscles of the hands and feet occurring in paroxysms.

Ætiology.—It occurs at all ages, but is especially frequent in infants and in young adults. In children, males are more often attacked than females; in older people the reverse holds good. In children, rickets and diarrhœa are predisposing causes; in women, pregnancy and lactation; and in adults of both sexes exposure to cold, recovery from febrile diseases, and the existence of dilatation of the stomach. It has occurred after the operative removal of the thyroid body; and similar paroxysms have been seen as the result of ergotism and in association with osteomalacia.

Symptoms.—There may be some discomfort or malaise, or stiffness in the arms, or tingling for some hours or days before the attack. Sometimes the paroxysm comes on suddenly without warning. The hands are then bent on the wrist, the fingers are flexed at the metacarpo-phalangeal joints, extended at the phalangeal joints, and pressed closely together with the thumbs bent into the palm of the hands; so that the fingers form a cone. The elbows are slightly flexed, and the arms are adducted to the sides. Sometimes the four fingers are flexed into the hand, the wrists extended, and the elbows fully flexed. In the lower extremities, the foot is extended on the leg, the tarsus arched, and the toes flexed and crowded together. These are the characteristic contractions, and in most cases these alone occur. In very severe cases spasm affects the muscles of the abdomen, chest, face, and tongue, as well as those of the back, causing slight opisthotonus, and of the eyes, causing strabismus. There may be some cramp-like pain in the parts affected; the back of the hands may be tumid, and the veins swollen. There may be sweating, flushing, and slight rise of temperature. The spasm ceases in from five to fifteen minutes, or it lasts one, two, or more hours; it gradually subsides, and recurs again after an interval of some hours or days.

In the intervals the nerves and muscles show an increased susceptibility to mechanical irritation (Chvostek). Percussion of the nerves causes contractions in the corresponding muscles, and stroking the face from above downwards causes contraction of the muscles one after the other. Trousseau first showed that in the intervals a fresh paroxysm could be brought on in a few minutes by firmly grasping the arms, or by pressure on the nerves and arteries. The motor nerves also are unduly susceptible to both faradism and galvanism (Erb). Very weak galvanic currents applied to them cause contraction of the muscles. ACC is most readily obtained, and the contractions to all currents are prolonged: AO tetanus and KO tetanus, observed in no other condition in man, may occur here. The sensitiveness of the sensory nerves to pressure and electrical currents is also increased (Hoffmann).

But there is not always an interval between the paroxysms. In infants a continuous spasm is more common, and in adults the spasm may not entirely relax; so that this form is called *remittent*, while the form with complete intervals is called *intermittent*.

The disease lasts from a few days to a few weeks, and recovery is the rule. But death may occur from exhaustion when the paroxysms are severe; or from pneumonia as a result of interference with respiration; or in infants from the diarrhœa which caused the disease. Occasionally some weakness of the legs remains for a short time after recovery; and muscular atrophy and fibrillary tremors have been seen.

Pathology.—*Post-mortem* examination has not revealed the cause of the disease. Its ætiology indicates a toxic, mostly autotoxic, origin; for instance, the possible absorption of poisons formed

in the fluids retained in a dilated stomach. Its distribution and the implication of the sensory, nutritive, and electrical functions suggest that the peripheral nerves are the parts upon which the toxins are acting (Judson Bury).

Diagnosis.—The distribution of the spasms—namely, their occurrence chiefly in the hands and arms—distinguishes it from *tetanus*. *Hysterical* contractions may assume the form of tetany; they are generally unilateral, and are associated with other hysterical conditions.

Treatment.—Bromide of potassium should be given in full doses, and the predisposing condition of the patient should be as far as possible removed. Thus, gastric dilatation should be treated promptly by lavage, or surgical operation (gastro-jejunostomy); in children, diarrhœa should be treated, and rickets met by cod-liver oil, iron, suitable diet, &c.; women should give up nursing their children, and should take iron and other tonics. Chloral, Indian hemp, Calabar bean, and morphia have also been given with more or less success. Chloroform inhalation stops the spasms for a while. Galvanism with the anode on the nerves may also be tried.

HYSTERIA

By hysteria is meant a disorder of the nervous system, which leads to various functional disturbances, sensory, motor, or visceral, of varying duration, and curable, though often very obstinate. The disturbances may be numbness or pain, paralysis, spasm, or general convulsions, flushes, palpitation, retention of the urine, and numerous other allied conditions. They occur from time to time in the same individual, and are usually spoken of as manifestations of hysteria, or hysterical symptoms or attacks; while the name hysteria is rather reserved for the general condition of the nervous system which is the cause of these disturbances, or forms the tendency to their occurrence. A person who has once had an attack, or a symptom of the kind called hysterical, is believed to be liable to further attacks, and practically to be one affected with hysteria.

The name hysteria (*ὑστέρα*, the womb) was given with the idea that the disease originated in a disorder of the uterus. But although sexual relations may have much to do with many cases of hysteria, this is not always so, even in adult females; while the disorder occurs in males, in quite young children, and in patients to whom such an explanation does not in the least apply.

Ætiology.—Hysteria affects chiefly females between the ages of fifteen and fifty: but it occurs in older women, and not infrequently in quite young girls. Adult males rarely, but boys more often, become subjects of the disease. Heredity has an important influence; hysteria is likely to appear in the offspring of hysterical parents, as well as in the children of the insane, of habitual drunkards, and of the sufferers from other neuroses. The hysterical

tendency is further fostered by bad moral training in the child such as allows it to yield to every emotional impulse, makes it expectant of undue sympathy in all its slight complaints, and does not teach it to be independent in battling with the troubles of everyday life.

Hysterical attacks are brought on by mental and physical disturbances of all kinds. The most frequent are emotional: domestic or business anxieties, grief for the loss of friends or relatives, serious quarrels or a mere difference of opinion, arrival at a rough part in the course of true love, are often the occasions of hysterical outbursts. Among physical causes we may have direct injury; thus, a blow on the stomach may cause gastralgia and flatulence, or an injury to the arm may be followed by hysterical paralysis or spasm. General illnesses, by their weakening effect, may cause various hysterical symptoms; or local lesions may lead to hysterical manifestations of the part primarily diseased. Thus a faucial or laryngeal catarrh may be followed by hysterical aphonia, or an actual synovitis by hysterical pain in the joint. Among the local causes of hysteria are diseases of the uterus—especially flexions of various kinds—and disease of the ovaries, viz., ovaritis. The former may be influential by acting through the emotions, perhaps to a greater degree than other local lesions; but the cure of the malposition by no means always cures the hysteria. In many cases of hysteria there is tenderness on deep pressure in the iliac region, and this has been ascribed to a tender ovary; but not always, it would seem, on sufficient grounds. Firm pressure at the same spot will also sometimes put a stop to a violent hysterical attack.

Symptoms.—These may be classified as mental, sensory, motor, vasomotor, and visceral.

Mental Condition of Hysteria.—An important feature of the mental state in hysteria is the instability and variability of all the mental faculties, emotions, will, and intellectual processes. Most apparent is the excessive development of the emotional faculties, and the deficient exercise of the will. The subjects of hysteria give way readily to grief, or joy, and seem to have little power of controlling the flow of tears, or the outburst of laughter. They are self-conscious, and show a constant desire for the sympathy of those around them. Thence arises a willingness to continue ill for the sympathy it excites, and in extreme cases the artificial production of symptoms and lesions by which their friends, and even their medical attendants, are led to believe them to be seriously ill. For instance, a girl will produce an artificial eruption on the skin by nitric acid, or cantharides powder, or the ends of matches; or will make a tumour in the face by accumulating substances between the gum and the cheek, or will show some foreign body that she states has formed in the rectum or vagina. Such performances one can scarcely doubt are intentional, but they are connected by every gradation with the quite involuntary simulation of disease, and occur, as a rule, in those who for other reasons may properly be regarded as hysterical. The intelligence, however, is often quite

good, but they have a deficient power of voluntary attention, or concentration upon a given object. And another characteristic, in part an outcome of the last, is a constant tendency to mental abstraction, so that they are absent-minded or dreamy.

Sensory Symptoms.—Of these may be noticed increased acuteness of the senses, amounting to intolerance of light or sound, or extreme hyperæsthesia. The patient will insist on the blinds being drawn, and complain of the least noise, or the slightest touch or shock. Tenderness is often noted at different parts of the body, especially the spine, the ovaries (as already mentioned), the left hypochondrium, the infra-mammary region, or the top of the head. Sometimes pressure in the one or other of these regions causes pain radiating from the spot, followed by *globus* (*see* p. 415), and even by a complete convulsive fit. Such spots have been called *hysterogenic*. Numbness and tingling, or other dysæsthesiæ, are occasionally felt. Sometimes sensation is lost in one or other limb, or in one half of the body, constituting *hysterical hemianæsthesia*. The loss of sensation is often strictly limited to one half of the body, ceasing sharply at the middle line, and involving the mucous membranes as well as the skin. In other cases, the anæsthesia does not reach to the middle line, but only affects the limbs, and side of the trunk: or it may involve the peripheral part of the arm or leg, or of both, the limiting line being perpendicular to the length of the limb (*segmental anæsthesia*), and the distribution differing materially from that of lesions of the peripheral nerves, or sensory roots (*see* p. 262). In others, again, the anæsthesia is disposed irregularly in separate areas (*disseminated anæsthesia*). Loss of sensation to pain is generally more pronounced than tactile or thermic loss: and pins may be thrust deeply into the skin without the patient's knowledge. A complete hemianæsthesia is accompanied by defects of the special senses of sight, hearing, smell, and taste on the same side of the body. The affection of sight is a crossed amblyopia; it involves the sharpness as well as the field of vision (which is contracted from periphery to centre), and the fields of the several colours (*see* p. 225). Sometimes the local application of metal plates will restore sensation in the affected part, but at the same time cause anæsthesia at the same spot on the opposite side of the body (*metallo-therapy*). This so-called *transfer* may be effected also in hysterical paralysis and contractions, and by other means such as large magnets, galvanic and static electricity, tuning forks, or sinapisms. It is no doubt a mental process. In a similar way the subsidence of any one symptom, whether sensory, motor, or visceral, may be succeeded by the appearance of another; and the cure of the latter by the reappearance of the first; or by the occurrence of a third.

Motor Symptoms. *Paralysis.*—Hysterical aphonia is not uncommon; the adductors of the vocal cords can be seen to be immobile (*see* Paralysis of the Laryngeal Muscles); but the fault is probably in the volition of the patient. Abductor paralysis,

existing alone, is very rarely the result of hysteria; it causes stridor and dyspnœa, with cyanosis, and anxious expression of face, and may even in hysteria lead to dangerous asphyxia. Dysphagia may arise from functional paralysis of the pharyngeal muscles. Ptosis also occurs as a hysterical symptom; it may be single or double. Paralysis of the limbs occurs in the form of paraplegia or hemiplegia; or all the limbs may be paralysed together. The paralysis in these cases is often not complete, and if the patient makes an effort to move the limb in a particular direction, it may be seen that some antagonistic muscles contract. The patient may assert that she is unable to lift the arm, yet if it is raised by any one else she will often keep it supported or let it drop only half-way, showing that the muscles believed to be paralysed have still a considerable amount of power. Also, if the attention is distracted to other things the patient may unconsciously move the supposed useless limb. If one lower extremity is alone affected, on attempting to walk the patient drags the paralysed limb behind her, making no effort to bring it forward, but only hopping along on the sound limb. The nutrition of the muscles and the electrical reactions are generally normal, but wasting of muscles is sometimes observed, especially in parts which are and have been affected by long-standing paralysis, as well as by contracture, or by anæsthesia. Knee-jerks are generally normal, and there is no true continuous ankle-clonus, but there is often a clonus lasting only a few seconds; and in some cases the knee-jerks are excessive. In paraplegia the legs can often be moved in bed, but the patient is quite unable to stand, and there is never incontinence of urine or fæces; in hemiplegia the leg is sometimes worse than the arm, and the face and tongue are always spared. Paralysis is sometimes, but not always, accompanied by anæsthesia. In a rare form of hysteria, every attempt to move, or contract a muscle, is painful (*akinesia-algera*). Another form of hysteria is a disorder called *astasia-abasia*, in which the patient can neither stand nor walk, though he can move the legs in bed, and there is neither inco-ordination nor sensory failure.

Ataxia.—Occasionally a well-marked ataxia is present in hysteria, either alone or associated with paralysis.

Tonic Contractions.—A common manifestation of hysteria is the tonic contraction of one or more muscles, often for long periods of time. Such contractions may come on after recovery from hysterical fits, and they may be excited directly by a blow or by emotional disturbance. The arm or the leg, or both together, are sometimes affected; the arm is generally flexed at the elbow, and drawn close to the side; the leg is rigidly extended. The limbs resist any effort to change their position, and the muscular contraction is stronger the greater the external force applied. It does not relax even in sleep, but only in the deepest chloroform narcosis. Rigidity of both legs is not common; when it occurs it is mostly in the position of extension. Contraction of all four limbs is very rare.

Trismus, or closure of the jaw, and laryngeal spasm are other forms of hysterical contraction.

Contractions may last months or years, or they may suddenly cease under the influence of emotion, or of the application of the faradic current.

Clonic Contractions.—These may occur in the form of tremor, of different degrees of rapidity in different cases: or of more decided rhythmical movements, such as nodding or rotatory movements of the head, or spasms of the muscles at the back of the neck, producing a spurious torticollis (Gowers). Or one fist may be constantly beaten upon the opposite hand, or the knee or thigh; or the shoulders may be constantly lifted and depressed. Some movements have a rather close resemblance to ordinary chorea, and have been called hysterical chorea; they are generally more sudden, more regular and rhythmical, and less like combined movements. The extensive movements of the whole trunk in bowing and swinging, which have been described as *chorea major,* are mostly of a hysterical nature.

The Hysterical Fit.—The attacks known as hysterical convulsions and "fits of hysterics" are commonly caused by emotional disturbance. Nevertheless they may occur in the middle of the night. An attack begins with *globus hystericus,* a sensation as if a ball were rising in the throat, threatening to choke the sufferer; with this there is giddiness, or palpitation, and the patient may burst into a fit of crying, or of uncontrollable laughter. In other cases after the globus the patient falls to the ground, or on to a chair or sofa, and at once passes into convulsions. These may at first be of a tonic kind; the body and legs are rigidly extended; the body arched forwards in a state of opisthotonus, with perhaps only the head and heels touching the ground; the arms are rigidly extended, either close to the body or at right angles to it, and the hands are clenched. The movements that succeed are of the most varied description; often they have every appearance of being made with a purpose. The back of the head may be repeatedly dashed against the floor until it actually bleeds; the limbs are thrown wildly about, and the bystanders are struck, or clutched at; if the limbs are restrained the struggling and fighting become more violent. Sometimes the patient gnashes her teeth, and may groan or shriek. The eyelids are generally closed, and resist attempts to open them; if they are opened, the eyeballs are rolled upwards under the upper lid. The face is usually red, and not livid as in epilepsy. There may be some saliva issuing from the mouth, but the tongue is not, as a rule, bitten. Consciousness is not entirely lost; the patient does not answer questions, but her actions may be guided by what is said in her presence, and, as already stated, there is automatic resistance to those who restrain her. After the active struggling movements have continued some minutes they commonly cease, and the patient lies panting, with closed eyes, muttering, or delirious, not responding to the appeals of her friends, until she again goes off into convulsions. These alternations may be repeated for two or three

hours. Recovery is often quite rapid; the movements cease, the patient opens her eyes and looks round, wonders what she has been doing, or, recognising it from former experience, may burst out crying. Headache may be present for some time afterwards, and a recurrence of the attack is not infrequent within a few days. The patients state that they have no knowledge of what has happened.

Charcot and his school used to describe a very severe form of hysterical attack (*hystero-epilepsy*, *hysteria major*). It was preceded by various minor troubles, changed manner, hallucinations, nausea, yawning, palpitation, anæsthesia, &c., and then passed through a regular series, consisting of an epileptic period; a period of contortions and great movements, especially pronounced opisthotonus; a period of attitudes indicative of the feelings of joy, grief, terror, &c.; and finally a period of delirium. A modern French writer, however, states that nobody now describes the attack of hysteria as Charcot did: and it was always remarked that such extreme conditions were rarely observed in England or in the United States.

Visceral and Vasomotor Symptoms.—Globus hystericus has been already mentioned, and dysphagia; the latter may be due to spasm of the œsophagus, which generally yields to steady pressure with the bougie, if this should be required to distinguish it from organic stricture. Vomiting, gastralgia, and flatulent distension are common accompaniments of hysteria. Anorexia may be a marked feature, and food may be refused for long periods; often in these cases some deception is practised, and food is taken secretly. The so-called fasting-girls are usually hysterical individuals whose ailments are fostered by the sympathy and ignorant wonder of the public, and the love of gain of their immediate friends. One variety of this form of hysteria is the *anorexia nervosa* of Gull, in which extreme emaciation may take place. It is often associated with excessive activity, the thin, delicate girl being constantly on the move, and walking several miles every day. The refusal of food may carry emaciation beyond the point of possible recovery, and several cases have been fatal. On the side of the circulation we may have palpitation, flushings, rapid or slow pulse, cardiac pain or pain like angina, syncope, and local pallor or mottling of the skin. Erythromelalgia and angio-neurotic œdema also occur in hysterical persons. The respiration may be extraordinarily rapid, and reach 70, 80, or 90 in the minute without any pulmonary lesion; the patient is able to go about without much distress; and during sleep the respirations fall to 20 or 18. Hysterical cough is common; it is generally incessant and noisy, or "barking" in character. Constant hiccough lasting for hours or days may be a hysterical feature. The urine passed after a hysterical fit is generally abundant, pale, and of low specific gravity. A diminution of secretion, *ischuria*, occasionally occurs. Retention of urine is not uncommon in hysteria, incontinence is rare; it is often said that hysterical women never

wet their beds. Similarly, constipation occurs, but never incontinence of fæces, and rarely diarrhœa. Elevations of temperature to 110°, 116°, and even 122° Fahr. have been recorded from time to time, which can only be explained on the view that they are related in some way to hysteria. They may occur in patients ill of other diseases, such as phthisis, and may be sometimes the result of compression of the bulb of the thermometer, or the application to it of hot flannels or poultices on the part of the patient. The temperature is rarely the same in different parts of the body; or at different times within the same hour. But a true *hysterical* or *neurotic pyrexia* undoubtedly occurs, as a fever continuous over some days, or as slight occasional rises to 102°, 103°, or 104°.

The following conditions are closely related to hysteria: Catalepsy, trance or lethargy, narcolepsy, somnambulism, and double personality.

In *catalepsy* the limbs tend to remain for long periods in whatever position the observer likes to place them. The patient appears to be deprived of voluntary movement; when another tries to lift the limb there is at first some resistance, then it yields, and if placed in a new position it remains so for a long time; ultimately, however, the limb will yield to gravity, and gradually sink into a position in accordance with it. The peculiar nature of the resistance to passive motions has led to the term "flexibilitas cerea." Catalepsy is often accompanied in hysteria with disorders of sensations. Besides forming a part of hysteria, catalepsy may occur after weakening illnesses in those who have shown no other indications of hysteria, in some mental affections, and occasionally in meningitis and apoplectic coma. It may also form a part of induced hypnotism.

Trance or *lethargy* is, like catalepsy, sometimes the result of hysteria, or of exhausting illness, or of hypnotism. The patient is in a peculiar condition resembling sleep, and may remain so for days or even weeks. The face is pale, the limbs relaxed, the eyelids resist efforts to open them. The pupils are moderately contracted or dilated, and react to light. The pulse is small, the heart-sounds are feeble or inaudible, and the breathing is extremely quiet, so that occasionally the patient has been thought to be dead. In prolonged cases there are remissions in which the patient may take food, relapsing again into stupor. Most cases recover.

Narcolepsy, or the occurrence of short attacks of sleep, beginning suddenly, lasting a few minutes to one or two hours, and ceasing suddenly, is seen in hysteria, but also in neurasthenia, and epilepsy.

In the milder forms of *somnambulism* the person rises up from the bed without waking, and walks about the house, with eyes open and staring, not regarding things around him, yet avoiding obstacles, and returning to bed without waking. In the morning he has no recollection of what has occurred. In other cases, a series of acts are performed in reasonable sequence, obviously prompted by an idea or the memory of some particular event, but with the same unconsciousness of surroundings not related to the act.

Cases of *double personality* are rare. As an instance may be mentioned the young lady, who without warning fell into a sound sleep for some hours, and on awaking was found to have lost all memory of reading, writing, and cyphering, and of persons and things about her. This knowledge she had to acquire laboriously again; but some time later she again fell asleep, and woke to find herself in her original condition or first state, with complete ignorance of what had occurred in her new, or second, state. Later again she relapsed into this new state, knowing then only so much as she had learned on the previous occasion. And so for some years she changed from state to state; the life of the first, third, and fifth periods was continuous, but different from the continuous life of the second, fourth, and sixth periods, and she had in the one personality no memory or consciousness of the events of the other.

In some individuals three or more personalities have been assumed.

Hysteria has some close relations with insanity; many insane women have previously been hysterical, and it is not always easy to draw the line between the two states.

The Nature of Hysteria.—Explanations of hysteria are various, and by no means satisfying. It is important for the student to understand clearly that hysteria is not *shamming* or *malingering*, though it may resemble it closely, and though in any given patient there may be a gradual transition from the condition of a genuine hysteric to that of a wilful deceiver. The patient with hysterical paralysis, or spasm, is not wilfully representing herself as unable to move the leg when she really can; she is at the time totally unable to supply the voluntary power necessary to move the limb, and if a sudden recovery should take place from excitement, the action of a galvanic battery, the threat of some disagreeable proceeding; or, what is more common, if she should slowly recover, from the steady moral pressure of encouragement and the absence of unnecessary sympathy, this is because the will is suddenly or gradually stirred up to exertion by fresh motives or impressions upon the judgment or emotions. It is, indeed, not so much the will to do wrong as the absence of will to do right. The absence of will and the predominance and variability of the emotions have been long regarded as the essential features of the disease; but it is quite obvious that various sensory, visceral, and vasomotor manifestations are beyond the influence of the will.

For some, hysteria is only a form of insanity. Babinski regards it as a disease by suggestion; only such symptoms as can be produced or removed by suggestion are hysterical. Janet looks upon it as a form of mental depression characterised by retraction of the field of personal consciousness, and dissociation of the mental processes and symptoms which ought to lie in close union. Acts may be performed and sensations recorded in a subconscious area, when the conscious area, which controls it, is in abeyance.

Freud's hypothesis is to the effect that hysteria depends on a painful

reminiscence which is not entirely dismissed from the mind, but is dissociated from the idea, and is transformed into a certain physical —that is, the hysterical—manifestation, while the idea is forgotten. The physical manifestation will remain until the patient can be made to see that it is the outward expression of the original forgotten idea. In Freud's opinion, the painful reminiscence is always one of a sexual nature; but even if the theory is true, this last proposition would be difficult to accept.

Diagnosis.—This depends chiefly on the age and the sex, which have been already specified; on the fact of previous manifestations; and on the history of the commencement of the present complaint, which is often sudden or occurs after a fit or other hysterical symptom, or after an emotional disturbance or blow quite inadequate for the production of an organic lesion. The absence of organic disease is in favour of, but its presence does not disprove, hysteria. Hysterical disorders are variable in degree from day to day, and are often less pronounced when the attention is distracted to other things. Further, the patient may suffer from different symptoms successively, the first recovering as the later ones appear. Some disorders are in themselves characteristic, such as—globus hystericus, aphonia from adductor paralysis of the larynx, and hemianæsthesia. The distinctive points about the paralysis are, especially, the variability from day to day, the unconscious contractions of antagonistic muscles when the attempt is made to move a limb, and of the muscles supposed to be paralysed when the patient's attention is withdrawn. The diagnosis of hysterical from epileptic fits has already been discussed (*see* p. 386). But true epileptic fits are sometimes succeeded by hysteroid convulsions.

Prognosis.—The sufferer from hysteria almost invariably recovers from her symptoms or attacks, though it may be after a very long time in cases where their nature is not recognised; and with advancing age the disease itself dies out. The possibilities in the other direction are that death might occur from exhaustion of hysterical vomiting, or anorexia; or from asphyxia in the rare abductor paralysis. According to Charcot prolonged hysterical contracture may be followed by lateral sclerosis of the spinal cord.

Treatment.—Both the general tendency to hysteria, and the particular form it takes in the case before one, have to be considered; and in either case the treatment may be both moral and physical. The patient should be placed under the best hygienic conditions. Fresh air, good food, moderate but not exhausting exercise, relief from mental overstrain or worry, the use of blood-tonics, such as iron, and the condition of the bowels, should all be considered. In the pursuance of moral treatment, it may be necessary to remove the patient from her friends, whose ignorant sympathy only prolongs the morbid condition. Many patients recover at once in hospital, where the sympathy they get is no more than is in proportion to the danger of their case. Success has often attended the use of the Weir-Mitchell method, in which the patient is not only

removed from her friends, but is absolutely isolated, and visited only by her nurses and the physician, until some improvement has been made. She is at the same time kept entirely at rest, fed abundantly, and submitted to the operations of massage and faradism. The former process directly favours the flow of lymph and venous blood in their respective vessels, and the latter, by contracting the muscles, improves their nutrition, and assists the effect of massage upon the vessels. The moral influence of the physician should be in the direction of encouragement to do things that appear impossible, of promise that recovery will come with patience and perseverance, of no undue sympathy for her in her ailments, even the reverse of sympathy in some of her symptoms such as vomiting, and of no excessive attention on his part to the symptoms themselves, by which she may be led to magnify their importance. At the same time, it is essential not to allow her to suppose that he regards her as shamming or malingering, as her confidence in him will then be lost.

In addition to general tonics, some drugs known as anti-spasmodics have a beneficial effect in hysteria. They are musk, asafœtida, valerian, and valerianate of zinc.

Passing now to the particular symptoms, we may treat hysterical pains with local applications such as belladonna and fomentations, though the attention should not be fixed too much upon them, but rather upon the general condition. Internal sedatives should, if possible, be avoided. Anæsthesia can sometimes be cured by the wire-brush electrode of the faradic current. The treatment of hysterical aphonia will be described under Diseases of the Larynx. For the various paralyses of the limbs, such as hemiplegia and paraplegia, the application of a strong faradic current is of much value. Sometimes a single application is sufficient; more often the paralyses require, in addition, a great deal of moral assistance. The patient must be assured that if she tries she will find herself not so weak as she thinks, and that she will gain strength day by day; where the leg is paralysed, she must be put upon her feet every day between assistants, and induced to make an effort to walk. Similarly with the arm; this must be raised for her, and she must be shown that it is not entirely helpless. In course of time she will gradually gain confidence in her powers, and often show a genuine satisfaction at her improving condition. Tonic contractions may cease on the application of galvanism, or of a circular blister round the limb, or after frictions with liniments, or passive extension. In bad cases it may be necessary to put the patient under chloroform, straighten the limb, and fix it to a splint. Where hysterical convulsions occur frequently, the anti-spasmodics should be given in combination with the general treatment mentioned above. Bromide of potassium is of little value unless the fits are partly epileptic. When the hysterical fit is coming on, it may be sometimes prevented by the use of some diffusible stimulant like ether, or ammonia, or by the strong will of another, forcing the patient to the exercise of her own will. The fit, once developed, can

generally be stopped by very strong impressions upon the senses; such as dashing cold water over the head and face, slapping the face and chest with a wet towel, applying strong ammonia to the nostrils, closing the mouth and nostrils for a few seconds, so as to cause a deep inspiration, or by deep pressure in the ovarian region. Gowers recommends in troublesome cases the injection of $\frac{1}{10}$ or $\frac{1}{12}$ grain of apomorphin subcutaneously. Hysterical vomiting and anorexia are especially suited to the Weir-Mitchell treatment; in anorexia the feeding must be abundant and frequent; in vomiting, food may be supplied by the rectum, and by the nasal tube. The discomfort caused by both of these methods no doubt contributes to their success.

NEURASTHENIA

This term is now in frequent use to designate conditions which mostly result from exhaustion of nervous power. These may arise in men as well as in women, and do so especially in those who are the subject of hereditary influence, or have been submitted to prolonged mental and physical strain, alcoholic or sexual indulgence, or business anxieties. Such persons find themselves after a time unequal to any physical or mental exertion, and suffer from various symptoms, such as oppression at the top of the head, frontal or occipital headache, weakness of the eyes, loss of sleep, and giddiness; loss of appetite, indigestion, constipation of the bowels, with sallowness and some loss of flesh, deficient cardiac power, and hence cold hands and feet; or it may be pain in the back, with tenderness at one particular vertebral spine (*spinal irritation*), pain radiating down the limbs, numbness, or tingling, &c. These, as will be seen, are so many functional disturbances which may be induced, some in one case, some in another, by a want of proportion between bodily income and expenditure; they scarcely constitute a separate disease. Those who suffer from these troubles are not necessarily hysterical; indeed, they present some important differences. Hysteria occurs almost exclusively in women; neurasthenia is more frequent in men. Hysteria occurs in those who have but little to do; neurasthenia in those who are overworked mentally or physically. The hysterical woman looks for sympathy; the patient whose nerve-power is exhausted often conceals his deficiencies and strives to overcome them.

Treatment.—Rest is the chief, as it is obviously the most natural, treatment; and it must be both bodily and mental. Good and abundant food must be given, and the bowels regulated; stimulants, narcotics, and anodynes as a rule should be avoided. Recovery is often assisted by the Weir-Mitchell method (*see* p. 419); but massage of itself may be a useful adjunct to bodily rest, without the moral advantage of isolation. Rest may be combined with suitable hydropathic treatment; and this may be supplemented by change of air and scene.

MASSAGE

Massage, which renders great service in hysteria, neurasthenia, and various other complaints, is the systematic manipulation of the limbs and other parts of the body. In the cases in which its use has been advised, it is mostly the muscles that are manipulated, as, for instance, in paralysis, contractures, hysteria, neurasthenia, &c. The methods of manipulation include friction, pressure, and percussion of the limb, and passive movement of the joints.

Friction, or *effleurage.*—The part is stroked with the palm of the hand in an upward or centripetal direction, and the hands are used one after the other with regularity, and more or less quickly according to circumstances.

Pressure includes *pétrissage,* by which a portion of muscle is picked up with the fingers and thumb of one or both hands; it is subjected to firm pressure and rolled between the fingers and the subjacent tissues. Other portions are similarly treated one after another, the operator working, as in the previous exercise, from periphery to centre. *Malaxation* is manipulation with the whole hand. *Pincement* is pinching of the skin. *Foulage* is deep pressure with the tips of the fingers, often in a circular or oval direction.

Percussion includes *tapotement,* or percussion with the tips of the fingers, the palmar surfaces of the tips, the palm of the hand, the back of the half-closed hand, the ulnar or radial border of the hand (*hachure*), or the whole hand hollowed so as to enclose some air between it and the surface of the limb; and *flagellation,* or striking with a wet towel.

The joints may be subjected to movements of circumduction, flexion and extension, and traction. Rubbing movements should be centripetal; they should be done with the dry hand, without the intervention of oil, ointments, or liniments. The duration of massage should be from five to fifteen minutes on any one occasion; but in recent cases the sittings may be three or four in a day. The effect of these manipulations is to promote the flow of lymph and blood in their respective vessels, and to stimulate the muscles of the skin and the skin reflexes.

HYPOCHONDRIASIS

This is a mental disorder, which in its mildest form comes frequently under the notice of the general physician, and only in extreme cases requires the same special care as is given to the insane. It consists in a morbid anxiety on the part of a patient as to his own health, and a morbid magnification of his sensations, so that he imagines illnesses that do not really exist. It is more common in men than in women, and most frequent between the ages of twenty and forty. There is sometimes a hereditary taint

of insanity from the parents; and the complaint may be originated by various depressing circumstances, such as business anxieties, moral considerations, the existence of gout, or slight digestive disturbances.

The sufferer from hypochondriasis is constantly under the impression that he is the subject of serious disease; every sensation that he has contributes to this idea, and he can turn his attention seriously to nothing else. He scrutinises with the utmost care his tongue, the colour of his skin, or the consistence and colour of his motions, and magnifies every abdominal sensation into a wearing or acute pain, which must be, according to him, due to cancer, or to internal ulcer, or to some other serious disease of which he has heard; whereas a most thorough examination fails to reveal anything at all, or at most some trifling disturbance of the stomach or bowels. In a large number of cases the complaints have reference to the abdomen; but there are other patients whose only concern is their sexual functions. They are frequently unmarried men between the ages of twenty and thirty-five, who have mostly led chaste lives, but may have masturbated when younger. Occasional pollutions at night, and the escape of a little prostatic secretion after defæcation, lead them to believe that their "vital fluids" are draining away from them; they complain of weakness, giddiness, oppression on the top of the head, inability to attend to their business, loss of memory, and shyness in presence of the other sex; they are convinced that they are impotent, that their complaint can be read in their faces, that they can never marry, and that their future is ruined. Sometimes hypochondriasis takes the form of a morbid dread of syphilis (*syphilophobia*) in one who has exposed himself to the risk, but has never had the slightest indication of infection; every sensation about the genitals, or ache or pain in other parts of the body, is put down to the dread disease, and no assurances to the contrary have any effect. In other cases cranial sensations are the prominent feature, and the sufferer fears tumour of the brain or madness.

Hypochondriasis may last for years, with intervals of improvement. Occasionally, it results in definite insanity of melancholic type; but in its ordinary forms it must be distinguished, if possible, from melancholia. The true hypochondriac does not have delusions, and is not suicidal. The diagnosis of hyochondriasis rests upon the manner of the patient and the nature of his complaints, combined with the absence of all serious disease, which should, of course, be carefully searched for. Lesions may sometimes be present which will not, however, account for the intensity or abundance of the symptoms.

Treatment.—This is in great part moral; the ailments must not be entirely ignored, nor must their importance be confirmed by too numerous prescriptions; the patient must be induced to turn his mind to other matters, to associate with those who may distract his attention from dwelling upon his own health, or to seek variety

in travel or other healthy recreation. Any defect in health, such as anæmia, constipation, indigestion, or the gouty state, should receive appropriate treatment.

NEURALGIA

This term is used for a special kind of pain, felt in the course of a particular nerve and its branches, and apparently of a purely functional character. It should not include the pains which may arise in a nerve in consequence of a lesion of its trunk, by such structural changes as, *e.g.*, the pressure of a tumour, or the existence of neuritis. But, even with the exclusion of these, it is obvious, as Head shows, that a number of pains described as neuralgia are pains referred to the peripheral nerves, as a result of visceral disease; and these, therefore, if functional so far as the painful nerve is concerned, are organic in their origin.

Ætiology.—Neuralgia is a disease of early adult and middle age, being most common between twenty and sixty. It is somewhat more frequent in women than in men. A neuropathic disposition is said to be an important disposing cause: patients are nervous, excitable, or have a family history of insanity, hysteria, epilepsy, or other nerve complaints; but Head denies this as regards pure trigeminal neuralgia. Some toxic conditions, such as rheumatism, gout, alcoholism, lead-poisoning, malaria, influenza, and diabetes, are antecedents of neuralgia. The exciting agents are depressed health from any cause, such as deficiency of food, over-lactation, and especially anæmia; physical fatigue, depressing emotions, and exposure to cold, such as the direct incidence of a draught of cold air upon the nerve concerned.

Symptoms.—The pain of neuralgia is deep-seated, and corresponds pretty closely to the position of a nerve-trunk, spreading along its course or radiating with its branches. It is accordingly often one-sided, but it may be bilateral, and even symmetrical. In character it is variable—shooting, stabbing, boring, burning, gnawing, or throbbing. It comes on in paroxysms, lasting a few minutes to an hour or more. Even in the shorter periods, the pain varies much in intensity; in the interval there may be complete freedom from pain, or at most a dull aching. The attacks may recur frequently in the same day; and their recurrence may be periodic, *e.g.*, lasting the whole day, and absent at night, or *vice versâ*. The *tender points* of Valleix are spots on the surface of the skin, which are tender to firm pressure—they lie in the course of the affected nerve or its branches, and correspond to the point of exit of the nerve from a bone, or where it perforates the fascia, or where it passes over a hard surface, or where the nerve divides into two branches, or where two nerves anastomose. Head thinks that these are not limited to true neuralgia, but that they represent in most cases the superficial tender areas of visceral referred pains.

Such *visceral referred pains* are due to definite lesions of visceral organs, and are characterised by superficial tenderness of the skin over areas which correspond not to the distribution of peripheral nerves but to the sensory nerve-roots, and to the successive segments of the spinal axis, as shown, so far as the neck, trunk, and limbs are concerned, in Figs. 26 and 27 (*see also* pp. 258, 259, and 262). This form of tenderness is best elicited, and its extent mapped out, by lightly pinching up successive portions of the skin, or by the pressure of a small rounded body, such as the head of a small pin, or the rounded point of a pencil. In each of these areas a maximum point may be found, which is tender sooner or lasts tender longer, than the rest.

Occasionally some muscular spasm takes place as a reflex effect in the region of the nerve affected with neuralgia, and vasomotor disturbances may be present, such as pallor at the beginning of the attack, followed by flushing, sweating, lachrymation (in trifacial neuralgia), and œdema. The hair may change colour, or fall off, or, more rarely, it grows in excess.

Some forms of neuralgia may be more fully described.

Neuralgia of the Fifth Nerve (*Trifacial or Trigeminal Neuralgia; Prosopalgia; Tic Douloureux*).—This may affect either one branch of the fifth, or two of its branches; or the whole of the sensory division of the nerve.

When the *first* division is affected, the pain is over the forehead, the anterior half of the scalp, the eyelid, eye, and side of the nose (*supra-orbital neuralgia* or *brow ague*). Tender points are found at the supra-orbital notch, at the outer side of the eyelid, or at the side of the nose, and sometimes there is an *ocular* point within the eyeball.

If the *second* division is attacked the pain extends over the cheek between the orbit and the mouth, and to the ala of the nose. Tender points are found at the infra-orbital notch, the side of the nose, on the prominence of the malar bone, and along the line of the gums below it.

If the *third* division is affected, the pain spreads over the parietal eminence, the temple, the ear, the lower jaw, and the tongue. The chief tender points are over the dental foramen, and over the auriculo-temporal branch at the back part of the temple, or just above the zygoma in the front of the ear.

The pain is often exceedingly intense, lasting but for a few minutes, and recurring at regular intervals. It may radiate from one branch of the nerve to the other, or to some other nerve. In the most severe cases, the facial muscles are seized with spasm during the height of the pain (*tic douloureux*), and the vasomotor symptoms, such as flushing, local sweating, lachrymations, discharge of nasal mucus, and salivation, are well marked. Increased sensitiveness to sounds, and flashes of light in the eyes, may also occur. The attacks are also brought on by cold, and especially by mastication, so that in some cases feeding becomes most difficult.

A *cervico-occipital* neuralgia occurs with pain in the region of the

upper four cervical nerves, and over the back part of the head. Tender points are found where the great occipital nerve becomes superficial, in the posterior triangle over the brachial nerves, and over the parietal eminence; the last is common to this and trigeminal neuralgia. Occipital neuralgia may be excited by disease of the teeth. It is often bilateral, and the pain is more often continuous with exacerbations than truly intermitting.

Cervico-brachial and *brachial* neuralgia occur with pain extending over the area of distribution of the brachial plexus, and the tender points are most commonly in the axilla, at the posterior border of the deltoid, behind the elbow (superior ulnar), and in front of the wrist (inferior ulnar).

Intercostal neuralgia is generally more or less continuous with acute exacerbations; the pain takes the course of an intercostal space, and tender points are found near the spine, in the mid-axillary line, and near the middle line in front.

Lumbo-abdominal neuralgia corresponds to the lower dorsal nerves, and occupies the lower half of the trunk. Tender points are found in positions corresponding to those just mentioned—namely, near the spine, at the middle of the iliac crest, and at the lower end of the rectus muscle. A *scrotal* (or *labial*) point may also be found.

A *crural* neuralgia in the region of the supply of the lumbar plexus is rare. There are some painful affections of the foot, as *painful heel* and Morton's *metatarsal neuralgia*. The latter occurs especially in women, and consists of a cutting or burning pain at the metatarso-phalangeal joint of the fourth toe, which is brought on by walking, and may extend to the rest of the foot, to the calf and to the knee. It is attributed to lateral compression of nerves by the heads of the bones; but it may be a true neuralgia.

Sciatica, commonly regarded as a neuralgia, is nearly always a neuritis (*see* p. 250).

Diagnosis.—This rests chiefly upon the remittent and intermittent character of the pain, and the absence of other symptoms indicating any organic lesion of the nerve, or of other parts connected with the nerve. In neuritis the pain is more continuous, and the nerve-trunk is tender in its whole length, and not only at points of emergence from deeper structures: long-standing inflammation, or compression by new growth, causes persistent anæsthesia, atrophy of muscles, or lasting trophic changes. In the absence of these, a very long duration would be in favour of neuralgia and against organic change. In all cases the evidences of diseases competent to produce pain in the region of the nerve affected should be carefully sought for. The lesions likely to produce this effect vary, of course, in the different parts of the body. Disease of the bones and periosteum, and deep-seated tumours, may involve the main branches of nerves. The cervical and brachial nerves are involved in caries and new growths of the cervical spine. The brachial nerves may be wounded, and are often the seat of neuritis. Intercostal pains may be due to disease of the ribs, caries and

cancer of the vertebræ, spinal meningitis, tumours, and aneurysm. Disease of the lumber vertebræ causes pains in the area of the lumbar plexus, and sciatic pains are produced by disease of the sacro-iliac joint, and of the hip joint, by psoas abscess, pelvic tumours, and tumours of the femur. Tabes dorsalis produces its shooting and stabbing neuralgic pains, which are generally bilateral, and are sooner or later accompanied by other distinctive symptoms. Among visceral referred pains may be mentioned especially pains due to carious teeth, glaucoma, errors of refraction, disease of the ear, some forms of heart disease, and renal calculus.

The **Pathology** of pure functional neuralgia is as yet unknown. Sclerosis, with destruction of nerve elements, has been found in some long-standing cases; for instance, in the Gasserian ganglion after excision. This perhaps only confirms our knowledge that neuritis causes neuralgic pains, and does not explain the acute and more transient forms.

Treatment.—This includes the removal of the cause if it can be ascertained, the improvement of any general ill-health which may excite or predispose to it, and the use of such drugs or methods of treatment as may modify the morbid condition of the nerve or centre. As we are not using the term "neuralgia" for the pains of neuritis or nerve-compression, the *removal of the cause* can only apply to those sources of irritation, such as carious teeth, by which a referred pain is produced. Though this may be effected, it does not follow that the neuralgia will at once cease; the altered condition of the nervous structure still counts for something, and the pain may be only lessened until other remedies are called in to assist. This fact should make one very cautious in recommending the removal of teeth, unless it is certain that the neuralgia is wholly or chiefly dependent upon the offending member. Many patients have submitted to the loss of all the teeth on one side of the jaw without any material benefit. The *general treatment* of the patient consists mainly in the administration of good food, especially fat-foods, and of tonics, such as cod-liver oil, quinine, iron, nux vomica, and strychnia. Among the remedies which are especially *directed against the local disease* are the following: Arsenic, in full doses (5, 7, or 10 minims of the liquor), or small doses gradually increased, especially in malarial cases; ammonium chloride (15, 20, or 25 grains three times daily); potassium bromide, in full doses; butyl-chloral hydrate (5 or 10 grains), especially in neuralgia of the fifth nerve; aspirin (10 grains); antipyrin or phenacetin (10 grains); tincture of gelsemium (15 minims); and exalgin (1 to 3 grains). Cannabis indica, ether, valerian, turpentine, and nitro-glycerin are sometimes useful; and a small dose of brandy or wine will often give much relief, but it is obvious that its use should be indulged in with much caution, since drinking habits may easily result from it under those circumstances. The same may be said, with a similar caution, of opium, morphia, and cocain, of which the last two may be given by subcutaneous injection, morphia in a dose of $\frac{1}{12}$ to

$\frac{1}{6}$ grain, cocain in a dose of $\frac{1}{2}$ to 1 grain locally. Trousseau, however, gave increasingly large doses of extract of opium for the violent pain of the worst cases of tic douloureux. Relief may also be obtained by external treatment, such as the application of various anodyne liniments (aconite, belladonna, opium, or chloroform), of menthol, of capsicum ointment or capsicum pencil (Girol), and of the ointment of veratria or of aconitia. Counter-irritation may be applied by mustard or blisters; and the actual cautery has been used, especially in spinal neuralgia. Success has sometimes attended the injection into the nerve itself or one of its branches, of 1 to 4 minims of a 2 per cent. solution of osmic acid, or of 5 to 10 minims of alcohol (80 per cent.).

Electricity is sometimes of benefit. A strong current, either faradic or galvanic, acting as a sort of counter-irritant, does good in some recent cases of hysterical neuralgia; more often a weak current seems advisable to act as a sedative to the nerves. For this a strength of from 2 to 5 milliampères should be used, with the anode on the seat of pain and the kathode in some other part of the body, the direction of the flow being a matter of no importance. A very weak faradic current may serve the same purpose.

Finally, in extreme cases, when all other methods have failed, neurotomy, neurectomy, or nerve-stretching offer themselves as possible means of recovery. In the severest forms of trigeminal neuralgia the Gasserian ganglion has several times been removed with a good result.

WRITERS' CRAMP AND ALLIED NEUROSES

Those persons whose occupations necessitate complicated movements for long periods of time, such as clerks, pianists, violinists, telegraph operators, cigar-makers, and others, may be subject, when engaged at work, to spasmodic and irregular contraction of the muscles concerned, so that the movement is badly performed and ultimately cannot be effected at all. A large number of those who thus suffer have previously had some organic or functional nervous disorder, or may be referred to the class of neuropathics by heredity, or of neurasthenics. The exciting cause is some depressing mental condition, mental anxiety, or business worry; an injury, or local disease of the hand or fingers; but more than all an excessive use of the hand in the occupation concerned.

The disease is most common in those who have a great deal of writing as their daily occupation, such as lawyers' clerks, secretaries, &c. It is hence called *writers' cramp* and *scriveners' palsy; graphospasm* and *mogigraphia* have been used as technical terms. This form is naturally more frequent in men than in women, and occurs mostly between the ages of twenty and forty. Gowers points out that in the act of writing the pen may be moved across the paper in four different ways—(1) The little finger is fixed on the paper,

and the fingers carrying the pen work upon the little finger as a pivot; (2) the wrist is fixed and acts as the pivot; (3) the pivot is at the centre of the forearm, resting perhaps on the edge of the table or desk; (4) all the movements take place from the shoulder. In the first method the movements of the fingers are most complicated and strained; and in the last there may be no finger movements at all. He states that writers' cramp scarcely ever affects those who employ the last two methods of writing.

Symptoms.—The affection generally comes on gradually; it may be felt at first as some degree of aching or strain, which is relieved by ceasing to write. After a time the act of writing is accompanied by a spasmodic tonic contraction of the finger or thumb holding the pen; the finger is pressed firmly on the pen, or it is flexed so as to move up the pen, or it slips off the pen so that the latter is grasped between the fore and middle fingers. The thumb may be similarly affected, or the fingers may be extended or lifted from the paper, or the pen may be driven into the paper, or the hand stops its movements entirely. The attempt to continue writing under these conditions produces a cramped, irregular, angular writing, with thick down-strokes; and after a time the spasm becomes so pronounced as to render the act impossible. This is the *spasmodic* or *spastic* form of Benedikt, which is by far the most common; but sometimes there is tremor of the fingers —*tremulous* form; and a *paralytic* form with fatigue alone has been described, but is quite rare. The spastic form often leads, by the frequent contraction of the muscles, to pains in the hands and wrist, which may after a time become distinctly neuralgic in character; and there is often some tingling or sense of numbness. The spasm may be limited entirely to the act of writing, and other movements, even of a delicate nature, can be performed without difficulty. Sometimes writers' cramp is associated in the same person with spasm on playing the piano or violin, and not infrequently in severe cases some other operation may be at the same time imperfectly performed.

The muscular power is for the most part preserved, or there may be a little weakness of grasp, or slight but definite weakness of certain muscles of the hand (Poore). The electric reactions may be quite normal, or they show a slight increase or diminution of irritability in some old cases.

The course of the disease is variable. In slight cases treated at once by perfect rest from writing, the patient may recover completely; but if he has persevered, forcing himself to write by steadying his hand with the other, or by mechanical contrivances, and has ignored all treatment, the disease is often quite obstinate, and may never be thoroughly cured.

The **Diagnosis** is not generally difficult; writers' cramp at least is not likely to be mistaken for anything else, but it must be remembered that some nervous diseases, such as chorea, hemiplegia, and other paralyses involving power in the hand, may be first detected

in the attempt to write, and may be regarded wrongly as writers' cramp. Nervous people, too, who have obtained some acquaintance with the disease may easily fancy that a little fatigue is the commencement of it.

Pathology.—This affection has been thought to arise from the weakness of certain muscles, and the over-action of antagonist muscles; or from weakness of one muscle being supplemented by another muscle, which in its turn gets fatigued and is followed by another, until all are worn out; or as the result of reflex action, stimulated through the sensory nerves. A probable explanation is, that it is due to a defect in the centres associated for the act of writing by a morbid lowering of resistance in the commissural connections between the centres, so that there is a radiation of impulses, and so over-action of muscles not necessarily engaged in the act.

Treatment.—The first essential is complete rest from writing. In mild cases this is sometimes sufficient to effect a cure in one or two months. Gowers then insists that, on again beginning to write, the patient should learn to write from the shoulder entirely. In more severe cases a much longer rest is required, and if writing is necessary to the patient, he may learn to write with the left hand or use a type-writer. Occasionally, but by no means always, the newly-educated left hand also becomes affected. Various devices have been invented, or are improvised by the patients themselves, to save the strain on the muscles of the fingers, such as running the pen through a cork, which gives a larger grasp: or holding a wooden ball in the hand, upon which the pen is fixed at the required angle. Nussbaum's "bracelet" carries the pen, and surrounds the fingers, so that they hold it by muscles (abductors) different from those commonly employed in writing. But, as a rule, these instruments only postpone the time at which complete rest must be taken. A return to the normal condition of nerve and muscle-action may be sought in the use of general and nervine tonics, such as iron, quinine, arsenic, and strychnia; and in local treatment, such as electricity, gymnastic exercises, passive manipulations, and especially massage. The first is recommended in the form of a continuous current with the anode stationary upon the brachial plexus, or upon the peripheral nerves and muscles concerned, and the kathode on the cervical spine.

The treatment of the other occupation-neuroses must be the same in principle as that already described for writers' cramp.

DISEASES OF THE MUSCLES

MYALGIA

(*Muscular Rheumatism. Rheumatic Myositis*)

This name is given to a painful affection, apparently involving the muscles or fasciæ. The connection with true rheumatism is not always obvious, nor is it even certain that the muscles or fasciæ are really involved; but the disorder is often the direct result of damp or cold, or of excessive muscular exertion or strain. Possibly toxins, or poisons absorbed from the alimentary canal, are responsible for some of the pains which occur in muscular or fibrous tissues. This is at any rate certainly true of muscular pains in the acute infectious diseases, such as small-pox, relapsing fever, typhus, and others. Recently the terms *fibrositis* and *fibro-myositis* have been used to indicate the affections, hitherto called rheumatic and apparently seated in muscles, tendons, or fasciæ; or in the fibrous tissues about joints. The terms are more precise, but their pathological accuracy has still, perhaps, to be proved.

Symptoms.—As a rule, only one muscle or group of muscles is affected at a time; and as certain muscles are particularly prone to it, special names are given to the disease, according to its locality. The symptoms are intense pain on attempted movement involving the muscle, and tenderness on manipulation. The pain comes on rather suddenly, and when it is severe necessitates the patient assuming a position by which it can be relieved; and this leads sometimes to a true reflex contraction of the muscle. A slight degree of pyrexia may accompany the illnesss, but it is more often absent.

The more usual seats of the disease are the following: (1) Lumbar and lumbo-spinal muscles—*lumbago.* This is common in advanced life, and in men more than in women. The patient walks with difficulty, and in a stooping position; any movement of the lumbar region is painful. (2) Intercostal muscles—*pleurodynia.* Breathing, coughing, and all respiratory movements cause severe pain, so that pleurisy may be suspected. But there is no rub, and the constitutional disturbance is slight or none. (3) Cervical muscles —*rheumatic torticollis,* or stiff neck. (4) Muscles of the shoulder—*omalgia.* (5) Muscles of the scalp—*rheumatic cephalalgia.*

Treatment.—Complete rest is desirable, and benefit is derived from local applications, such as hot poultices and fomentations, belladonna and aconite applications. Subcutaneous saline injections (*see* p. 251) are also recommended here. Massage is of value, and probably assists in dispersing any effusions in the fibrous tissues.

Free perspiration should be induced, by means of the vapour or Turkish bath. Both the galvanic and faradic currents are useful. Internally the most useful remedies are potassium iodide, in full doses; salicylic acid in doses of 15 or 20 grains; sodium salicylate; aspirin; antipyrin. Cataphoresis or ionic medication, with iodide of potassium, or iodine and carbonate of lithia, applied to the painful part is also recommended. As a strained position, unconsciously assumed, is sometimes a cause, this should be considered: and avoided in the future.

MYOSITIS

Inflammation of muscles is rare as a primary disease. General myositis, or *polymyositis*, occurs in various forms. In *dermatomyositis* the muscles of the extremities, and later those of the trunk, are swollen, œdematous, stiff, and painful on movement and pressure. The skin over the affected muscles is œdematous, and presents erythematous, erysipelatoid, or eczematous patches. There is moderate fever, and the spleen is enlarged. The cases last for a few months to two or three years, and are often fatal from implication of the muscles of deglutition and respiration, or recover with atrophy of the muscles. The muscles are found to be swollen, yellowish-white in colour, soft and friable. The muscular fibres are swollen and granular, or hyaline and waxy, or contain vacuoles; and the connective tissue is infiltrated with leucocytes and œdema-fluid. The cause is entirely unknown, but is probably microbic.

An allied affection is *hæmorrhagic polymyositis*, in which bleeding takes place into the inflamed muscles, petechiæ occur under the skin, and the patient has palpitation and tachycardia.

Myositis is sometimes associated with multiple neuritis (*neuromyositis*), and this may be seen in chronic alcoholism (*alcoholic myositis*). The muscles are painful and tender, and become hard and shrunken; while the skin over them may be œdematous. Under the microscope are found irregular areas of cirrhotic induration, and more acute cellular infiltration of the perimysium. A chronic "rheumatic" myositis has also been described, affecting mostly the muscles of the head and neck, but also those of the gluteal, abdominal, lumbar, and deltoid regions. In early cases the muscles are found to be swollen, in older cases there are thickenings or indurated masses like cartilage. There may be tenderness, as well as sharp and cramplike or aching pains. Their occurrence in the temporals, occipito-frontales, or upper parts of sternomastoids or trapezius muscles is accompanied by paroxysms of severe headache, and the condition has been described as *indurative headache*.

Secondary myositis occurs in various infectious diseases (*infective myositis*), producing a diffuse swelling and infiltration of the muscle; or abscesses may form, as seen in pyæmia (*metastatic myositis*),

septicæmia, glanders, typhoid fever, and malignant endocarditis. Trichiniasis, described below, is essentially a parasitic myositis.

Tubercle and *syphilis* also involve muscles. The former occurs as a metastatic deposit secondary to tubercle of the bones, glands, or viscera; or arises by direct invasion from adjacent parts. Syphilis produces a diffuse myositis in almost any stage of the disease, and in the tertiary stage the typical *gumma*. A local inflammation of the muscles sometimes occurs in the neighbourhood of the joints in rheumatic fever and gonorrhœal synovitis.

Myositis ossificans is a disease in which the muscles are converted into osseous tissue. It always begins in early childhood, invades first the muscles of the back, and runs a progressive course. A *myositis fibrosa* has also been observed.

Treatment.—The indications for treatment are the relief of pain, when present, by local applications, or in severe cases by morphia and other sedatives; the removal of the cause, as in alcoholic cases; and specific treatment, as in syphilis, when potassium iodide should be of use. Limited lesions in tubercular, septic, and metastatic myositis may require incision and antiseptic methods. Indurative headache and the associated myositis are benefited by massage, and by such drugs as sodium salicylate, aspirin, and antipyrin.

PARASITIC DISEASES OF MUSCLE

The muscles may be invaded by certain animal parasites. Of these the *trichina* or *trichinella spiralis* is the most serious, as it sets up a polymyositis, which is often fatal. *Echinococcus*, or *hydatid cysts*, and the *cysticercus telæ cellulosæ* are also found in muscle, but cause very little, and that only local, trouble.

TRICHINIASIS

The disease, known also as *trichinosis*, is due to the nematode worm above named, which is found in enormous numbers in the voluntary muscles throughout the body. The disease is rare in England, but is not uncommon in Germany. The worms are parasitic in rats, and are in some way conveyed by them to pigs, being deposited by thousands in their muscles. Within twenty-four hours of the ingestion of such pork by human beings, sexually mature trichinæ are found in the intestine. As usual, the females are more numerous and larger; they measure from one-twelfth to one-seventh of an inch, while the males are from one-twentieth to one-fourteenth of an inch, and differ from the females in presenting two small processes at the tail. Within seven days after ingestion, embryos are formed within the ova, and are discharged from the females already hatched. According to the view more generally accepted the females bore their way into the villi and other parts of the mucous membrane, and the embryos are deposited in the

lymph-vessels or chyle-vessels, by which they are carried ultimately to the voluntary muscles. In the muscles they increase in size, and possibly move about in the course of the muscular fibres. About the second week they reach the full size corresponding to this stage, namely $\frac{1}{25}$ inch, or a little less, and two or three weeks later they become coiled up, and, as a result of the irritation which they produce, are gradually surrounded with a capsule. This is oval, or rather fusiform with an oval bulging in the middle, and lies always parallel to the muscular fibres; it measures $\frac{1}{75}$ inch in length by $\frac{1}{130}$ inch in breadth. It is at first nucleated and transparent, but afterwards becomes calcified, especially at its ends. Calcification in the human subject probably does not take place under twelve months, and even then does not interfere with the life of the parasite within. Indeed, it may remain in this condition for years; or it may perish, and be converted into a structureless mass. The muscles in which the trichinæ are deposited acquire a pale reddish-grey colour, the fibres lose their striation, and become brittle and homogeneous, with numerous minute fissures. With the exception of the heart, all the striated muscles of the body may be affected, but the capsules are most abundant in the diaphragm, the intercostal muscles, the biceps, and the muscles of the larynx and of the throat. As long as the worm remains alive in its capsule, it has the power of developing into a sexually mature trichina on being taken into the stomach of a suitable host. Trichinæ are found not only in muscles, but in the connective tissue of other parts, especially the fat layers and coats of the intestine.

Symptoms.—These consist mainly of febrile reaction with local evidence of inflammation of the muscles. In some cases there are at first gastro-intestinal disturbances, such as epigastric pressure, nausea, vomiting, and diarrhœa, or perhaps constipation. But these are often slight, and the commencement is, like that of many febrile diseases, characterised by loss of appetite, sleeplessness, lassitude, and depression. Very soon the arms and legs become painful; the knees and elbows are either flexed or extended, but in each case any alteration of the position is extremely painful, and the patient avoids every movement. The muscles of the limbs are tender, and feel hard and swollen to the touch. The electric reactions of nerve and muscles are diminished; mastication becomes painful, and the jaws may be closed for weeks; the implication of the respiratory muscles causes shallow and interrupted breathing; and coughing, sneezing, and yawning are difficult or impossible. The inability to cough up the secretions aggravates the dyspnœa seriously. The movements of the eyeballs are painful, and the power of accommodation is said to be lost at the same time. Towards the end of the first week appears another symptom—namely, œdema. This occurs first in the eyelids, then the rest of the face and neck may be affected, and sometimes even the upper and lower extremities. Its causation is not clear. The fever is

seldom very high, or continuous; the temperature is generally below 102°, but may rise to 104°. The pulse is rapid, there may be profuse sweating, and a miliary eruption; and there are erythematous patches, wheals, or vesicles as in dermatomyositis, or petechiæ and pustules. There is leucocytosis, and the eosinophiles are very numerous, reaching in some cases 80 per cent. of the leucocytes. The tongue is dry, red, and slightly furred; sometimes there are headache and stupor. Death may take place in the fourth or fifth week, or earlier, from exhaustion, pneumonia, or bronchitis; and if the patient recovers, convalescence is slow, and hindered by muscular pains, muscular atrophy, and persisting œdema.

Pathological Anatomy.—The only characteristic change is the condition of the muscles. There are sometimes signs of hæmorrhagic catarrhal inflammation of the small intestine; the liver is often fatty; the spleen is not enlarged.

Diagnosis.—There is a certain resemblance between trichiniasis and typhoid fever, in the febrile reaction and diffused pains, but with the progress of the disease the differences become marked, especially the absence in trichiniasis of initial headache, rose spots, splenic enlargement, and the Widal reaction. Cases of trichiniasis occur in groups, since an affected animal is likely to be eaten by many individuals or a family. A suspicion may be confirmed by the examination of the fæces, or of a portion of muscle removed during life.

Treatment.—The trichinæ situate in the muscles are beyond our reach; we can only hope to destroy the parasites in the intestine. For this purpose castor oil or calomel in large doses may be given. Benzine, 1 or 2 drachms daily, in gelatine capsules; glycerine, a tablespoonful every hour or two; and picric acid, $\frac{1}{2}$ to 1 grain daily, have been recommended.

The muscular pains may be treated with narcotics internally and chloroform or belladonna externally.

NEW GROWTHS IN MUSCLE

The tumours found in muscle are *rhabdomyoma, fibroma, chondroma, osteoma, sarcoma, angeioma, lipoma, gumma* (*see* Myositis), and *carcinoma*. The last is frequently due to invasion from adjacent parts, as, *e.g.*, the pectoral muscles from cancer of the breast, the intercostal muscles from cancer of the lung, and the orbicularis oris from epithelioma of the lip.

MUSCULAR ATROPHY

(*Amyotrophy*)

Atrophy of muscular tissue takes place under a variety of conditions, and has been divided into *simple atrophy* and *degenerative atrophy:* in the former the muscular fibrillæ diminish in size, while in the latter they diminish in number as well. The two conditions are not entirely distinct in their origin—that is, the same cause may in one case produce the first, and, operating for a longer time, or more acutely, may bring about the second, severer form.

Simple atrophy is seen especially after acute or long illnesses, as a result of starvation, some kinds of intoxication, and locally from disuse, and from paralysis in cerebral lesions. In some chronic joint affections muscular atrophy occurs, which may be either simple or degenerative.

The degenerative variety is seen in the most pronounced form in the several lesions of the spinal cord and nerves which involve the lower neuron in one or other part, and hence bring about Wallerian degeneration in the periphery. These *myelopathic* and *neuropathic* forms have already been described (*see* Multiple Neuritis, Progressive Muscular Atrophy, Anterior Poliomyelitis, &c.).

There remain certain *myopathic* forms of atrophy, that is, primary atrophy not referable to any lesion of the nerves or nerve-centres. The origin of these cases is still very obscure; they show strong family or hereditary connections; they occur in young people, are more frequent in males, but are often transmitted through the females, and no doubt depend upon a congenital tendency to early degeneration. The muscles selected for first invasion are different from those picked out by spinal disease; sensation is unaffected, fibrillary twitchings are generally absent, and though the response to electrical currents is by no means ready, there is no reaction of degeneration.

Several varieties have been recognised: in some the muscle is in the early stage enlarged by fatty and fibrous tissue, constituting pseudo-hypertrophy, to which atrophy eventually succeeds; in others, there is atrophy from the first, and rarely, if ever, the semblance of hypertrophy. Erb groups them all under the term *progressive muscular dystrophy.*

PSEUDO-HYPERTROPHIC MUSCULAR PARALYSIS

The striking feature of this disease is the enlargement of the muscles with loss of power; but the same muscles subsequently become atrophied, and atrophy also occurs in some muscles which have never been enlarged.

Ætiology.—It is much more common in boys than in girls, often occurs in different members of the same family, and is handed down to succeeding generations, as a rule, through the mother, and

not through the father. But since a girl who develops the disease is little likely to become a mother, it is mainly through healthy mothers, the sisters of those affected with the disease, that it is transmitted. Thus, to quote Gowers, "a boy suffered, and his sister, unaffected, had two sons diseased, and a daughter free, of whose children two sons were the subject of the malady." It commonly shows itself in early childhood, either when the child first begins to walk, or when it has attained the age of five or six. In only a few cases it is delayed to the age of twelve or thirteen, and very rarely to eighteen or twenty.

Symptoms.—It may be first noticed as a delay in acquiring the art of walking, although the muscles of the limbs are sufficiently large, and apparently well developed: or, the child gets easily tired in walking, or walks unsteadily, and finds a difficulty in getting upstairs. As the child gets older, the condition of the muscles attracts attention. At first one calf, and then the other, is noticed to be large, and subsequently the enlargement may affect the glutei, the lumbar muscles, and those of the trunk, shoulder, and upper arm. The enlarged muscles are usually hard, firm, resisting, and suggestive of great strength, to which the actual capabilities of the patient by no means correspond. But this condition of apparent hypertrophy is not universal; generally some muscles are found to be atrophied, and these are mostly muscles in the upper part of the body, or some of those of the arm, especially the latissimus dorsi, the lower part of the pectoralis major, and the teres major; but the crural muscles are also often wasted. On the other hand, the deltoid and the infra-spinatus may be enlarged, but the muscles of the face, tongue, neck, forearm, and hands are either not affected at all, or affected very late. The weakness of the muscles produces some defects of motion, which are very characteristic. The patient has a waddling gait, the feet are widely separated, and the body is thrown from side to side with each step; the gait is further modified by the tendency to walk on the toes, which results from the diseased calf-muscles shortening and producing a modified talipes. In standing also, the legs are widely separated for the sake of equilibrium, and the back assumes the position of *lordosis*, or curvature with a deep lumbar concavity backwards. This is because the weakened glutei and extensors of the hip allow the pelvis to drop forwards, and the balance of the body is then only preserved by the shoulders being thrown back so that a line dropped from them falls even behind the sacrum. Another characteristic feature is the way in which the patient rises from the sitting position on the ground. He rolls over on his hands and knees and, if a chair or bed be near, pulls himself up by its means; but if alone, he lifts the knees from the ground so as to be on his hands and toes; then, swinging himself over towards one side, he places the opposite hand on its corresponding knee, and by its means straightens the leg. The same manœuvre is repeated with the other hand and knee, so that he now stands with legs

wide apart and a hand on each knee. With a great effort, then, the back is gradually straightened as the hands are brought higher and higher up the thighs.

The weakness of the spinal muscles is also shown by the inability to pick up objects from the floor, and by the falling forward of the body if the patient is sitting and leans forward too much; further, there is great difficulty in ascending stairs, so that the help of the banisters is sought.

The electrical condition of the affected muscles is not at first much altered. When they have become very weak there is diminution to both faradic and galvanic electricity. There is no degenerative reaction. The knee-jerk is, after a time, diminished, and in advanced cases lost.

Beyond this, the nervous system is normal: sensation is unaffected; the bladder is only affected occasionally towards the end; and the mental functions are mostly unimpaired, though, according to Ross, some degree of mental incapacity and even idiocy may co-exist.

After the first development of the symptoms the disease may remain stationary for two or three years, but the patient gradually gets weaker in the legs, the power of standing is lost, wasting takes place, especially in the muscles of the upper extremities, and the patient is confined to bed. Finally, death from intercurrent illness, such as inflammation of the lungs or an acute infectious disease, takes place about puberty in the majority of cases; but in those that have developed slowly the fatal termination may be correspondingly delayed.

Anatomical Appearances.—The abnormally enlarged muscles look to the naked eye like masses of fat, and under the microscope are seen to consist largely of adipose and fibrous tissue, with muscular fibres thinly scattered throughout. There is a large increase of the connective tissue between the muscular bundles, and fat then develops in this connective tissue. In the process of atrophy of the muscular fibres the transverse striæ become fainter, or are replaced by fatty granules, or by longitudinal striation. But some fibres remain normal in size and striation, though separated from one another by broad tracts of fat and connective tissue. Other fibres are diminished in size or irregular in diameter; and many have obviously disappeared. In some muscles the development of fat is not so marked, and the colour is better preserved, but there is a large excess of fibrous tissue.

The changes in the nervous system are slight and probably secondary: some diminution in the size and number of the anterior cornual cells, and in the number of nerve-fibres in the anterior roots, with increase of connective tissue in each case.

Diagnosis.—This is not generally difficult. The prominence of the calf muscles, associated with weakness, difficulty in walking and in rising from the ground, and lordosis, are commonly sufficient to distinguish it. Gowers lays more stress upon the enlargement

of the infra-spinatus together with wasting of the latissimus dorsi and the lower part of the pectoralis major. The *spastic paraplegia* of children may resemble it, as the calf muscles may be contracted, firm, and of good size; but this contraction is active and can be overcome, the knee-jerk is excessive, the muscles show a tendency to spasm, and the children do not rise from the ground in the way peculiar to pseudo-hypertrophic paralysis. In *spinal atrophies* the distribution of the order of wasting is somewhat different, and there is no enlargement at any time.

The **Prognosis** is very unfavourable, and **Treatment** can at best retard its progress a few months or years. Drugs probably have no influence, and electricity little, if any. Carefully planned gymnastic exercises have done good, and rubbing, massage, and passive movements may be of some use. When the gastrocnemii are so shortened as to prevent the patient standing, the tendons should be divided.

IDIOPATHIC MUSCULAR ATROPHY

Under this name are grouped some allied forms of disease in which muscular atrophy is the important characteristic, or exists alone. Like pseudo-hypertrophic muscular paralysis, idiopathic muscular atrophy occurs in several members of the same family, but it shows no preference for boys over girls. Weakness and wasting come on simultaneously, and are first noticed in some cases in the face, in others, in the upper extremities, and in others, again, in the lower extremities. Different types have been recognised by writers, but they are all probably allied to each other, as well as to pseudo-hypertrophic paralysis. It will be sufficient here to mention two of them.

Erb's juvenile form.—This commences usually about the age of puberty, and affects the muscles of the upper extremities first. The biceps, triceps, and supinator longus are atrophied, and the latissimus dorsi, the lower part of the pectoralis major, the serratus, trapezius, and rhomboid also suffer, but the deltoid, supra-spinatus, infra-spinatus, and subscapularis generally escape. The muscles of the forearms are but little affected, and the small muscles of the hand rarely. The spinal muscles vary in different cases; the intercostals, the diaphragm, and the abdominal muscles are sometimes affected. Of the leg-muscles, the glutei, the flexors of the hip, and the extensors of the knee are most often diseased, and the muscles below the knees often escape; but the peronei are atrophied occasionally. A temporary enlargement may occur in some muscles, especially the deltoid, infra-spinatus, and the muscles of the calves. If the disease begins in the legs, it is likely to be at an earlier age than when it begins in the shoulders and arms; and in either case the facial muscles rarely suffer.

Infantile form. Facio-scapulo-humeral type of Landouzy and Déjerine.—This begins at a very early age, and the facial muscles are first affected. The zygomatici fail early, the naso-labial fold

is lost, and in smiling the angles of the mouth are drawn outwards instead of upwards and outwards. The orbicularis oris is weak, so that the lower lip drops, the mouth "pouts," and the labials are badly pronounced. The orbicularis palpebrarum is occasionally affected, and the eyes remain unclosed; or the frontales are paralysed, and the forehead cannot be wrinkled. The tongue, the larynx, the pharynx, the muscles of mastication, and those of the eyeballs are never involved. Only later are the muscles of the shoulders, arms, hands, and perhaps of the legs, implicated.

In all these forms, as a rule, fibrillary twitchings are absent. Irritability is diminished equally to both electric currents, and there is no reaction of degeneration.

Deformities may occur similar to those of pseudo-hypertrophic paralysis, such as lordosis and talipes. The course of the disease is variable. It may remain limited to the muscles first affected, or at intervals of years it may spread to others. Its duration may be from ten to fifty years; and death takes place from intercurrent affections, such as phthisis. Hitherto only a few *post-mortem* examinations have been made. The muscles in one case were found to be small and pale, with intense granular and fatty degeneration of the muscular fibres; in another there was simple narrowing of the fibres. The spinal cord and nerves were normal.

Distal form.—Gowers and others have described cases in which the weakness involves first the movements of the fingers, toes, hands, and feet, and the corresponding muscles become atrophied; while the more proximal muscles of the thighs and upper arms are spared. In Gowers' case the sterno-mastoids and orbiculares palpebrarum were weakened, showing a resemblance to other myopathies: and in some cases examined *post mortem* the spinal cord has been found to be healthy.

Clinically these cases should be distinguished from the peroneal form of muscular atrophy, which is primarily spinal (*see* p. 295).

Treatment.—For the several forms of dystrophy electricity, massage, regulated voluntary exercise, mild gymnastics, and attention to the general health are recommended, in order to retard as much as possible the progress of the disease.

THOMSEN'S DISEASE

(*Congenital Myotonia*)

This very rare disease, of which hitherto only a few cases have been recorded in England, appears also to be a form of muscular dystrophy, and may properly be here considered.

It consists of a peculiar rigidity of the muscles, which comes on whenever they are called into contraction by voluntary impulses after a period of rest. Thus, if the patient wishes to walk and tries to rise from his seat, his muscles become rigid, and he is unable to move; the rigidity lasts a few seconds, and then relaxes

so that he can at length get up. His first few steps are attended with the same difficulty, but soon the contractions become more natural, and shortly the trouble ceases altogether, so that he walks with complete freedom and ease. If, however, he should stop for a minute, the muscles become rigid on his beginning to walk again. Quick movements are thus impossible immediately after rest, and sometimes accidents occur, as, for instance, when the patient is descending from a train, and having placed one foot on the ground, he is unable to bring the other out quickly after it, and falls in consequence. The muscles of the lower extremity are most affected, but those of the arms, and even those of the face, are sometimes involved to a less degree. The tendency to rigidity seems to be increased by cold and by mental anxiety. The following changes are noticed in the muscles: they are hypertrophied, but their power of contraction is rather diminished; contraction to direct mechanical stimulation (idio-muscular contraction) is said to be in some cases exaggerated; the closure contraction to strong galvanic currents is much prolonged beyond the time of application, and with continued application of the current a series of contractions has been seen to pass in a wave-like manner from the kathode to the anode. The reactions to faradism are often normal, but may be exaggerated in force and duration. The disease often lasts a very long time, but it may subside.

Ætiology.—It happens often early in life, may affect more than one member of a family, and is commonly regarded as being congenital.

Pathology.—In a case reported by Déjerine and Sottas, the patient died of nephritis. The muscles were large, protruding, tending to be more globular in shape, of a duller red colour, and less elastic than normally. The number and size of the muscle-nuclei were found to be increased, the fibres varied much in size, some being very large indeed, as if swollen. Sometimes the sarcous elements were separated and the sheath was filled with granules; or the muscle substance was degenerated and vacuolated. The connective tissue was not hypertrophied or fatty as in pseudo-hypertrophic paralysis. These results agree closely with what had been seen in portions of muscle excised during life. The muscle-nerves, nerve-trunks, spinal cord, and bulb were healthy.

Treatment.—Nothing has been found of use. Thomsen, who suffered from it himself, advised a life of continued activity.

MYOTONIA ATROPHICA

In this disorder stiffness of the muscles similar to that of Thomsen's disease is associated with muscular atrophy. The stiffness, or myotonia, is especially noticed in the inability to relax the flexor muscles after grasping strongly with the hand. The muscles affected by atrophy are the orbicularis oris and orbicularis palpe-

brarum (so that there is some resemblance to the Landouzy-Déjerine type of muscular dystrophy), the masseters, temporals, sterno-mastoids (severely), the muscles of the forearm, the vasti femoris, and the anterior tibial and peroneal muscles.

The disease begins generally in early adult life, and occurs in several members of the same family. Pathologically the muscles present overgrowth of connective tissue, with some degeneration of muscular fibres: and the most that can be found in the nervous system is some degeneration of the posterior columns of the spinal cord. More often the myotonia precedes the atrophy: sometimes the converse holds good.

AMYOTONIA CONGENITA

(*Myatonia Congenita*)

In this disease the muscles are extremely flaccid and wanting in tone, and the child lies about in any position and cannot sit up. The condition is noticed at or shortly after birth. Later it is unable to walk; all its actions are feeble, and passive movements at the joints are unusually free. The electric reactions are diminished and the deep reflexes are absent. In many cases there is slow and progressive amelioration; but others get worse. The disease may affect more than one member of the family; and in spite of the excessive hypotonus, the absence of local muscular wasting, the congenital occurrence and the acute onset in cases not congenital, they are probably to be regarded as belonging to the myopathic group.

FAMILY PERIODIC PARALYSIS

In this curious complaint, the patient suffers from attacks of gradually developing paralysis of the muscles of the trunk and limbs, which lasts on each occasion several hours and then completely passes off.

Ætiology.—This disease has also been observed in several members of a family, and is transmitted to the descendants of the sufferers. It affects the two sexes equally, and the first attack has generally occurred between the ages of six and twenty-four.

Symptoms.—The paralysis often begins at night. The muscles of the legs, then of the arms, and lastly those of the trunk and neck, gradually lose power, so that in the course of four or five hours the patient is quite unable to move his limbs. The weakness affects first the proximal parts of the limbs, and the distal parts later. The intercostal muscles are weakened, so that the breathing is shallow and feeble; and probably also the diaphragm is involved. The muscles supplied by cranial nerves are generally spared. The reflexes are gradually lost; and the reactions to both faradic and galvanic currents, which get less and less as the weak-

ness increases, are entirely lost with complete paralysis. Sensation and the mental state are unimpaired. After a few hours improvement begins: the muscles regain power in the reverse order of their loss of it, and after another six to twelve hours power is completely restored as well as the reflexes and electrical reactions.

The attacks at first occur at intervals of months, but they become more frequent until they may occur weekly or oftener: as middle age is reached they again become less frequent.

The **Pathology** is at present obscure. The probability seems to be that some toxin is operating upon the muscular fibres; and some interesting observations on the relations of the urine and of kreatinin-excretion to the attacks have been made.

Treatment.—Diuresis should be encouraged by salines, and mineral waters, in order to get rid of toxins.

MYASTHENIA GRAVIS

This is another disease profoundly affecting the action of the muscles; and in the absence of any constant change in the nerve centres or nerves, while lesions are frequently found in the muscles, it must be regarded for the present as a primary disorder of those structures, probably toxic in origin.

Symptoms.—The characteristic feature of the disease is weakness of the voluntary muscles, which are very rapidly exhausted by exertion, but recover their power after rest. In severe cases the weakness persists, and death often results either suddenly or with dyspnœa from respiratory paralysis. The muscles most frequently involved are those of the eyes, head, and neck, so that the patient has ptosis, diplopia, immobility of the face, difficulty of swallowing, defective articulation, and inability to support the head upright. But nearly all the muscles in the body may be affected: the patient may be unable to sit up, can only walk a few yards without stopping, or his respiration is impeded, and he has dangerous attacks of dyspnœa. In the limbs the proximal muscles are attacked more often than the distal. The condition is very variable in its intensity, and is aggravated by emotion, by cold, and by the menstrual function in women. The knee-jerk is generally active, sensory symptoms are seldom present, and the sphincters are not affected.

The affected muscles mostly react in a special manner to electrical currents—the *myasthenic reaction.* If the faradic current is applied to the muscles, they contract normally, but if it is continued, they soon become exhausted and fail to contract any further. If then the electrodes are removed, the muscle recovers, and then contracts well to the current, again becoming soon exhausted. Contraction to the galvanic current is persistent, and is scarcely at all affected by the length of application.

It affects the sexes equally, and occurs at all ages. Temporary improvement may take place, but a fatal result generally ensues.

Morbid Anatomy.—Several cases have now been examined *post-mortem.* In some the thymus has been persistent or enlarged, or the subject of lymphosarcoma; but in others it has been absent in accordance with normal conditions. In all of five cases examined by E. F. Buzzard, there were found collections of lymphocytes (lymphorrhages) in the muscles, and in some organs such as the thymus, liver, and adrenals; but the blood and lymph-glands were healthy.

Diagnosis.—It is likely to be mistaken for hysteria, diphtherial paralysis, and bulbar paralysis. From the latter it may be distinguished by the absence of atrophy in the muscles, by the myasthenic reaction, and the varying degrees of weakness.

Treatment.—Little can be done for it: the patient should be kept warm and at rest, and should be carefully fed and dieted. Tonics appear to have a little influence, and strychnine hypodermically is recommended. On the theory of autotoxic origin, intestinal antiseptics and irrigation of the lower bowel have been tried. Electricity and massage are not advisable.

DISEASES OF THE ORGANS OF RESPIRATION

PHYSICAL EXAMINATION OF THE CHEST

Since the lungs are contained almost entirely within the bony thorax, or chest, the diseases of these organs are likely to reveal themselves by modifications in the shape, in the movements, and in the acoustic phenomena yielded by the chest. A consideration of these various physical signs or indications of lung diseases must precede their systematic description.

For purposes of accurate description it is necessary to divide the surface of the chest into regions. The obvious divisions, front and back, left and right, are too extensive. But we can, with the help of certain readily recognisable structures, divide the surface of the chest into smaller areas. These structures are the sternum, the clavicles, and the nipples in front, the scapulæ behind, and the axillæ at the sides. The areas or regions named from them are the following: from above downwards in the middle line supra-sternal, upper sternal, mid-sternal, and lower sternal; on each side in front, supra-clavicular, clavicular, infra-clavicular, mammary and infra-mammary; under each arm, axillary and infra-axillary; behind, supra-spinous, infra-spinous, infra-scapular, and inter-scapular. Still greater accuracy is obtained by stating on which rib or intercostal space the point under investigation is situated, and how far from some fixed line or point, like the middle line, the edge of the sternum, the nipple, or the angle of the rib. Besides the middle line, the edge of the sternum (lateral sternal line), some other *vertical* lines are often employed, viz., the nipple, or mammary line; a mid-clavicular line dropped from the middle point of the clavicle, not quite identical with the nipple line; the parasternal line, midway between the lateral sternal and the nipple line; anterior axillary, mid-axillary, and posterior axillary lines; and a scapular line drawn through the lower end of the scapula. The ribs serve as horizontal guides, and they are best counted from the prominent ridge between the manubrium and gladiolus (*angulus Ludovici*), which corresponds to the second rib; and from the twelfth rib behind, which can be readily identified in most persons.

The modes of examination of the chest are inspection, including the use of Röntgen rays, palpation, mensuration, percussion, auscultation, and succussion.

INSPECTION

By looking at the chest in front, behind, and from above, any alteration in its shape and movements can be detected. The chief points to be noticed in a healthy adult chest are as follows: It has a somewhat flat oval form—that is, the antero-posterior diameter is much less than the transverse; its greater breadth is at the lower part; the clavicles are only slightly prominent, with but little depression above, and scarcely any below them; the position of the nipple is on the fourth rib, or on its upper or lower border; the angle (*epigastric angle*), which has its apex at the ensiform cartilage, and is bounded on each side by the seventh and eighth costal cartilages, is from 95 to 105 degrees; the scapula is closely adapted to the posterior part of the thorax; and the spine is straight. In inspiration the chest should expand from 2 to 3 inches in circumference, the two sides should move symmetrically, the epigastric angle should be widened, the sternum thrown forwards, and the lower ribs lifted; and there should be only very slight recession of the lowest intercostal spaces on deep breathing.

By inspection in disease we may see that one side is larger or more contracted than the other, that the movement is deficient on one or other side, or that the intercostal spaces are unduly sucked in; and we may also see at once the position of the heart, which may be disturbed by disease of the lung.

The deformities of rickets, and of angular and lateral curvatures, which are not due to diseases of the lungs, but may seriously impair the action of these organs, should be specially noted.

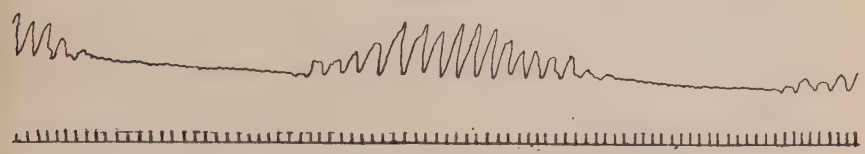
By inspection also, apart from changes in the shape and symmetry of the thorax, we may note the character of the respiratory movements. The normal frequency of respiration in adults is from 15 to 18 in the minute; in children it is much more rapid, and varies with the age. In different forms of disease, both pulmonary and of other origin, the movements may be slower or more rapid, shallower or deeper, feebler or stronger than is normal, and they may be irregular. The term *dyspnœa*, meaning difficult or bad breathing, is more generally used to indicate that the breathing is unduly rapid—shortness of breath—or that it is performed with effort. The necessity for such effort may lie in some obstruction to the passage of air, or in defective muscular power. The difficulty may be most marked during inspiration (*inspiratory dyspnœa*), or during expiration (*expiratory dyspnœa*). If the patient is obliged to sit up in order to breathe, as is the case in many pulmonary and cardiac diseases, the condition is called *orthopnœa*.

It is important also to note whether the breathing is effected more by the upper part of the chest, which is usual in women, or by the lower part and the diaphragm, which is more characteristic of men. The inspection must be extended to the abdominal walls, which reflect, so to say, the action of the diaphragm, advancing when it contracts and receding as it relaxes. A disproportionate use of one

part of the chest suggests disease in another part. The inspection should be made both during quiet breathing and when the patient makes a forced inspiration.

A peculiar form of breathing is known as *Cheyne-Stokes respiration.* It is characterised by alternating periods of very rapid and very slow movements. As the movements get slower there is at length a pause of several seconds (stage of *apnœa*); then the movements

FIG. 44



Cheyne-Stokes Respiration. The curve reads from left to right, and the time is marked below in seconds. The small undulations during the period of apnœa are due to the beats of the heart.

begin again, very feeble at first, then quicker and stronger, until they may occur at the rate of 50 to 60 a minute (stage of *dyspnœa* or *hyperpnœa*); then they quickly become slow again, and the cycle is completed by the long pause. The whole duration of a cycle may be from 20 to 60 seconds, and the number of respirations in a cycle varies from five to sixty (*see* Fig. 44). In some cases the changes in the breathing are accompanied by other phenomena. For instance, in the stage of rapid respiration the patient may become excited, groan, struggle, or even try to get out of bed; as the breathing slows he becomes quiet, drowsy, and apathetic. More often the pupils dilate in hyperpnœa, and contract again in apnœa. The pulse is often scarcely affected, but I have known it cease entirely for 30 seconds in the early and middle periods of hyperpnœa. Cheyne-Stokes respiration has not been fully explained, but it is attributed to diminished excitability of the respiratory centre with a defective supply of arterial blood. It occurs in many conditions, but most frequently in diseases of the brain and heart, in aneurysm, and in uræmia. It often precedes death only by a few hours or days; but it has been known to persist for months and even years; and it may subside entirely.

Biot's respiration is an allied condition, seen most commonly in meningitis: there are pauses of several seconds up to 30 or more occurring more or less periodically.

RÖNTGEN RAYS

This method of investigation is of great value in recognising the fact of disease in the chest, or in estimating its extent and position: the position and movements of the diaphragm, the presence of pulmonary consolidation, tubercle, new growths, and liquid effusions may be recognised by shadows visible on the screen; and of these photographs may be taken.

PALPATION

By this is meant the act of laying the hand upon the surface of the chest, either to test its movements or to study the vibrations of its walls produced by the voice or other cause. For the former purpose a hand is laid at the same time on each side below the clavicle, or in the infra-scapular or infra-axillary region, when the absolute and relative amounts of movement can be gauged with some accuracy. For the latter purpose the hand is placed flat upon the chest in different parts successively, and the patient speaks in a loud voice. In health the chest-wall is thrown into vibrations which are plainly perceptible to the hand laid upon it (*tactile vocal fremitus* or *tactile vibration*). For this it is necessary that there shall be a normal vibration of the vocal cords, and normal conductivity of the lungs with patent bronchial tubes and spongy lung-tissue. The amount of vibration differs in healthy people; it is greatest in adult males with deep sonorous voices; it is least, or it may be absent, in females and children. In disease it is diminished or abolished by anything which obstructs the bronchial tubes or compresses the lungs, so as to convert its spongy tissue into solid, *e.g.*, liquid in the pleural cavity. It is increased under some conditions of consolidation of the lung-tissue with patency of the bronchial tubes, especially pneumonia.

By palpation also can be recognised the vibrations of pleural friction, of bronchial narrowing (rhonchi), and of some sounds produced in cavities. The corresponding sounds are described under Auscultation.

MENSURATION

The chest may be measured in various ways. The ordinary tape measure gives the circumference, and if measurements be taken during expiration and full inspiration, the difference will give a rough idea of the expansion of the chest, or vital capacity. The tape should be applied opposite the nipples. By *callipers* the transverse and antero-posterior diameters can be estimated. The *cyrtometer* consists of two long pieces of soft metal, joined loosely together by one end of each. The point of junction is applied to the spine, and the metal rod on either side is wrapped round the side of the chest at any desired level, so as to take a mould of its shape or curve. The instrument is then carefully removed, without disturbing the moulded curve, and, if it is laid out on a large sheet of paper in the position it occupied while applied to the chest, a pencil can be traced round it, and a permanent record of the shape of the chest is thus obtained. The *perigraph* is another recording instrument invented by Graham Brown.

The movements of the chest-wall can be registered on a dial or paper by the *stethograph* of Reigel and the *thoracometer* of Sibson. The *spirometer* of Hutchinson records, in cubic inches, the air which is breathed out of the chest; the fullest possible expiration after a

deep inspiration gives the *vital capacity* (complemental, tidal, and supplemental air together), and this has been found to have a close relation to the height of the individual. It is on an average 174 cubic inches for a person five feet high, and rises 8 cubic inches for every inch of height. Waldenburg's *pneumatometer* measures the force of inspiration and expiration by means of a mercurial manometer. The inspiratory force raises from 70 to 100 mm. of mercury, and the expiratory force from 90 to 130 mm.

PERCUSSION

In percussion the chest is struck with the fingers or with an instrument called a hammer or *plessor*, so as to elicit a sound. In *immediate* percussion the chest is struck directly with the hammer or with the tips of the fingers, generally the fore, middle, and ring fingers, of the right hand. In *mediate* percussion a finger of the left hand or a small piece of ivory or other material, constituting a *pleximeter*, is laid upon the chest, and this is struck with the finger or hammer.

Percussion over the healthy lung elicits a sound, which varies in different parts of the chest, but which has the general characters of what is known as *pulmonary resonance*. It is a rather full and low-pitched note. It can be obtained on the right side from just above the clavicle to the upper portion of the sixth rib; over the whole of the sternum; on the left side from above the clavicle to the upper border of the fourth rib internal to the nipple, and outside the nipple down to the sixth rib, where it passes into the resonance of the stomach. In the right lateral region it extends from the axilla to a horizontal line cutting the eighth rib in the mid-axillary line; on the left side the axillary resonance is limited below by the upper border of the ninth rib. Posteriorly, the chest is resonant from the apices to the lower border of the eleventh rib on the left side, and to its upper border on the right side. The resonance extends a finger's breadth lower than these limits on deep inspiration. The fulness and loudness of the note are most marked in the second intercostal space in front and over the infra-scapular regions behind. Over the clavicle and sternum it is less full, and of higher pitch; and over the supra-spinous fossæ the note is often deficient, especially in very muscular or fat people. At its lower margin the pulmonary resonance is less marked, and approximates to the dulness of the parts below; it is called *transitional* dulness. This extends on the right side in a line above the liver, occupying the fifth space in front, and the seventh in the axillary line; on the left side the line runs from the sternum along the third space, turns vertically down within the nipple to the heart's apex, and may be again noticed in the eighth space just above the spleen. Round the heart this corresponds with the *deep cardiac dulness*.

The healthy percussion note is due to vibration of the chest-walls, and of the columns of air in the lung beneath the point struck.

The percussion note varies in healthy persons according to the thickness of the parietes (fat or muscle), and is modified in disease by alterations of the tissue of the lung.

The *intensity* of the note is diminished by solidification of the lung substance. There is then said to be impairment of resonance, or *dulness.* Much overlying fat or muscle will also diminish the sound on percussion. The intensity is increased by the lung-tissue becoming more open in structure and less finely spongy. This happens in emphysema, and the note is called *hyper-resonant.*

The *pitch* of the note is raised by an increase of tension in the chest-wall, by an increase of tension in the lung-tissue, and by a less length of the underlying air columns. It is of course lowered by the converse conditions. It frequently happens that diminution of intensity coincides with elevation of pitch, when the solidification of a portion of the lung shortens the columns of vibratile air under the part percussed.

A *tympanitic* or drum-like note is often observed very similar to that which may be obtained by percussing the distended stomach. This is a purer note than the normal percussion-note, and is due to vibrations taking place in a single large unbroken space. It occurs over very large cavities in phthisis, and in pneumothorax, where one pleural cavity is distended with air. A somewhat similar note, but higher pitched, is heard over the upper part of the lung in cases of pleuritic effusion occupying the lower half or two-thirds of the chest. It is known as *Skodaic resonance,* and is probably due to partial compression, relaxing the tissue of the lung, and thus giving it, so far as vibrations are concerned, some of the characters of a large continuous cavity.

If the finger is used as a pleximeter, the vibrations of the chest-wall can be *felt* at the moment of percussion; and a want of normal vibration, often called *resistance,* is readily appreciated.

AUSCULTATION

This is the study of the viscera or other parts of the body, by listening to the sounds that are produced within them. It may be *immediate,* when the ear itself is applied to the chest, either bare or with only a towel or handkerchief intervening; or *mediate,* when a sound-conducting instrument connects the chest of the patient and the ear of the listener. The instruments more commonly employed are (1) the *binaural stethoscope,* (2) the straight wooden or metal stethoscope, about seven inches long, and (3) the *phonendoscope,* in which the sounds are resonated. The first and third have the advantage of flexibility, and can be used in all positions of the patient.

By auscultation of the lungs we study the character of the breath-sounds, the transmission of the voice through the chest, and the transmission of the cough.

Auscultation of the Breath-sounds.—If the healthy lung is auscultated, one hears everywhere, with each respiration, a sound

which is known as the *normal breath-sound*, or *vesicular murmur*. It may be imitated by blowing softly, with the lips placed in the position to pronounce the German "w" or English soft "v." As its name implies, it has been regarded as due to vibrations produced in the air-vesicles, as the air passes from the minute bronchial tubes into the wider alveolar spaces beyond; but the vibrations of the air passing between the vocal cords certainly contribute to it. The vesicular murmur is heard during inspiration; but the expiratory act is either quite silent, or is accompanied by a similar sound, much softer, and much shorter in duration. In certain parts of the chest the vesicular murmur gives place to a sound having the characters which will be presently described as those of *bronchial breathing*. These parts are the upper end of the sternum, the first costal cartilages at their junction with the sternum, and a diamond-shaped space at the back in the middle line, including the seventh cervical and first dorsal spines. Elsewhere the vesicular murmur is always present, as long as the lung is healthy and the air-passages are pervious. In children the vesicular murmur is louder than in adults.

Diminished vesicular murmur, deficient entry of air, or absence of breath-sound, occurs if the air-vesicles are obliterated by pressure, or displaced from the surface of the chest, or if the bronchus communicating with them is obstructed or obliterated.

Increased vesicular murmur happens over both lungs from hurried respiration; over one lung or part of a lung, when another part of the lung is not properly in use. It is then called *compensatory* or *supplementary* breathing. It is louder and harsher than the normal breath-sound, and the expiratory murmur is almost or entirely suppressed.

Interrupted breathing.—In this the inspiratory murmur is jerky or wavy, from irregular expansion of the lung, of which the cause may be mechanical obstruction to the entry of air, irregular muscular action from nervousness, or the cardiac impulse. The term *cog-wheel respiration* is sometimes used.

Bronchial breathing or *tubular breathing*.—This modification of the breath-sounds has the following features: The inspiratory and expiratory sounds are of equal lengths; they are distinctly separate from one another; they are of higher pitch, and hollower than the vesicular murmur. The sound may be imitated by placing the mouth and tongue in the position to pronounce the German "ch," and then blowing in and out; but there may be very considerable variety in both the pitch and hollowness of sounds that may still all be called bronchial. Such double hollow sounds are heard normally over the larynx, over the trachea, and over the origins of the larger bronchi, at the top of the chest, as already stated; but if they are heard in other parts of the chest they are due to modifications of the lung tissue, mostly a conversion of the spongy lung tissue into solid lung tissue, either by filling up of the air cells (pneumonia, phthisis), or by compression from without

(pleuritic effusion). The necessary condition seems to be patency of the bronchial tubes with consolidation of the surrounding lung. Very different views are still held as to the cause of the sound. It is obvious that the vesicular element of the breath-sound is abolished. One of the earliest views was that the glottic sound was conducted to the surface by the solid lung; another view is that the glottic vibrations are more perfectly conducted along the tubes, dissipation being prevented by the solid lung (H. Mackenzie); another is that the glottic vibrations are resonated in the tube of the affected part; another, with which I am most in sympathy, is that the otherwise quiescent column of air in the bronchial tube of the consolidated part is set in vibration by the air currents moving across its mouth in the larger tube with which it communicates (Bullar). Bronchial breathing may also be produced in small cavities, and in dilated bronchial tubes. Its pitch and hollowness are determined by the length or size of the tube or cavity in which resonance takes place; the higher-pitched, "whiffing" varieties occurring in the narrower or shorter tubes, and the lower-pitched in the larger tubes.

Cavernous breathing.—By this term is meant a very hollow breath-sound, in other respects like bronchial breathing. Such hollow sounds often take place in rather large cavities, but there is no broad line of difference between cavernous breathing and hollow bronchial breathing; and breath-sounds which deserve the name of cavernous are often heard over solid lung.

Amphoric breathing.—This is a still more hollow, double sound with a peculiar metallic or ringing character, such as may be produced by blowing softly into the mouth of a narrow-necked glass jar or vase. In its strictest sense it is rarely heard, and then only in very large cavities, or in pneumothorax.

It is important to note that loudness is not a necessary feature of either bronchial, cavernous, or amphoric breathing; the latter especially is often quite soft.

Adventitious Sounds.—The word adventitious expresses the fact that these sounds are heard in addition to, and at the same time as, the ordinary breath-sounds or the breath-sounds modified as above. If they are not heard with tranquil breathing, the patient should inspire deeply, when they may become audible. The adventitious sounds are *rhonchi, stridor, râles,* and *friction sounds.*

Rhonchi are more or less musical sounds, due to obstruction of the bronchial tubes, by accumulation of mucus, thickening of the mucous membrane, or spasmodic contraction of their muscular fibres. The sounds vary very much according to the size of the bronchial tube and the extent of the narrowing, and are likened to various familiar sounds, such as cooing, groaning, snoring, grunting, or whistling. The lower-pitched, snoring sounds are called *sonorous* rhonchi, and are produced in the larger tubes; the higher-pitched, whistling sounds are called *sibilant* rhonchi, and are produced in the smaller tubes. They may be heard with expiration or inspiration,

and are constantly changing in position and loudness. Loud sonorous rhonchi are often audible to those standing near the patient, and constitute "wheezing."

Stridor is a loud, harshly musical sound, which is produced by constriction of the glottis, trachea, or one main bronchus. It is less changeable than rhonchus, is audible over the greater part of the chest, and can sometimes be heard by those near the patient without the aid of the stethoscope.

Post-tussive suction is the name given to a high-pitched sound, heard over a cavity (tubercular or bronchiectatic) during the inspiration following a cough; it is perhaps due to elastic recoil of the cavity-walls.

Râles are various forms of crackling or rattling sounds, which are produced in the medium-sized and smaller bronchial tubes, or in pulmonary cavities, by the air forcing its way into fluid secretions accumulated there, and thus causing bubbles to form and burst with a slight noise. They are sometimes distinguished as *moist* sounds, from rhonchi or *dry* sounds; but this is undesirable, if rhonchi may themselves be due to the presence of mucus. The râles differ according to the size of the bubbles, and are called *small, medium,* and *large.* Râles are also divided into *bubbling* and *crackling;* the latter have a sharp, clear, ringing, explosive character, which is probably due to their occurrence in the midst of consolidated lung, and to consequent special conditions of resonance; the former, or bubbling râles, are dull, not ringing or explosive, and occur mostly in tubes surrounded by normal spongy tissue. Crackling râles are sometimes called *consonating* from their supposed acoustic origin; and bubbling râles, *non-consonating* in contrast.

Thus we have râles which are

small, medium-sized, or large, } and at the same time { crackling (consonating), or bubbling (non-consonating).

Gurgling is a coarse râle which occurs in large cavities.

Crepitation is a term that has been used indiscriminately for all râles, but is now generally confined to a very fine râle, so fine as to be suggestive of an origin in dry materials (rubbing of hair close to the ear, rustling of silk, or tearing of paper). It is heard in the early stage of pneumonia, in œdema of the lung, and in lung that is forcibly expanded after prolonged collapse. It is probably due to the opening up of minute bronchioles, or even air-vesicles, which have been adherent by sticky fluid, or from simple disuse. Crepitation and the finer râles are heard only during inspiration; medium-sized and coarser râles may be heard during expiration also.

Metallic tinkling is resonance caused by a râle in a large cavity.

Friction-sound, or *pleuritic rub,* is produced by the rubbing together of two pleural surfaces roughened by inflammation. In its most characteristic form it is a rough, grating, interrupted sound like that which may be heard on forcibly dragging two pieces of leather over one another, or on rubbing the palmar surface of a finger over

a wooden surface. It is best heard during inspiration, but may be heard with expiration also. When it arises in the pleura which lies between the apex of the heart and the chest-wall, its loudness may be increased with each beat of the heart.

Auscultation of the Voice.—In most people the voice is transmitted through the chest, and can be heard by the ear or stethoscope placed on any part of it; this is called *vocal resonance.*

Diminished or absent vocal resonance.—In children and females with voices of high register, the vocal resonance may be slight or absent. In disease its absence is produced by obstruction of the bronchus, or compression of the lung, involving the bronchus.

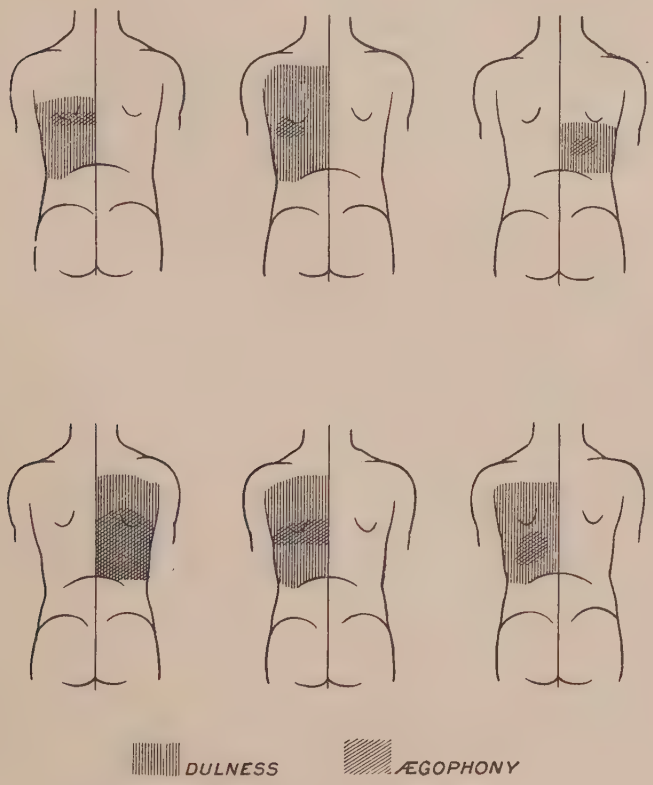
Increased vocal resonance. Bronchophony.—There is naturally a louder vocal resonance at those points where bronchial breathing is normally heard—namely, the sterno-clavicular articulation, and the inter-scapular region (*see* p. 451). In disease, it is caused by consolidation of the lung around bronchial tubes, such as occurs in pneumonia, tubercular consolidation, and sometimes compression by liquid. It is often regarded as the result of increased conducting power on the part of the solid lung, but the view that it is due to altered conditions of resonance in the tubes of the affected lung is, to my mind, more probable.

Pectoriloquy is the clear transmission of articulate sounds, as distinguished from mere loudness of transmission of the vocal vibrations in bronchophony. It may be recognised when the patient speaks loudly, but it is best observed by asking the patient to whisper, when the laryngeal vibrations are absent. It is observed over pulmonary cavities, and over consolidated lung.

Ægophony consists of a peculiar *nasal* or *twanging* modification of the voice, heard through the chest. It derives its name from its supposed resemblance to the bleating of a goat. It appears to be due to the suppression of the fundamental tone, and the lower harmonics of the vocal sounds, while the higher harmonics are transmitted in an accentuated form, and produce a discordant note. Its most common cause is undoubtedly the presence in the pleura of a liquid, by which the lung is compressed: and the bronchial tubes, in which, normally, full vocal resonance takes place, can in these new circumstances only *resonate* the higher harmonics. Ægophony is commonly heard at the middle of the back behind, internal to, or below, or over the lower end of the scapula; sometimes over a large part of the dull area with a vertical extent of several inches, and not necessarily limited to the upper edge of this area (*see* Fig. 45). It is occasionally heard in front. Sometimes, also, though rarely, it is heard distinctly when no liquid is present, though the condition of the tubes must be similarly modified by some other means; thus it may occur in pneumonic consolidation, with a bronchial tube containing fibrin. It is best brought out by asking the patient to utter words containing the vowels *i* and *e*, which depend on the presence of the higher harmonics, such as "three," or "ninety-nine."

Auscultation of the Cough.—The patient is directed to cough while the physician auscultates the chest. Increased resonance of the cough occurs under the same conditions as increased resonance of the voice (consolidated lung and cavity); moreover, the cough,

FIG. 45



Diagrams showing Areas of Dulness and Ægophony in Cases of Pleural Effusion.

and the forced inspiration preceding it, will reveal the existence of râles that are not obvious on ordinary inspiration. In infants the spontaneous cough supplies the information as to vocal resonance, which is given in adults by speaking.

AUSCULTATORY PERCUSSION

In this process a stethoscope is placed on the chest, and the surface is percussed around it; minute differences of resonance are thus detected. By a similar process the *bell sound* or *bruit d'airain* may be elicited in cases of pneumothorax. While the physician

listens with the stethoscope to one part of the chest, presumed to be the subject of pneumothorax, an assistant lays one coin on the chest, and strikes it with another. The noise is resonated in the hollow cavity, and transmitted as a loud ringing musical note through the stethoscope.

SUCCUSSION

In cases of hydro-pneumothorax or pyo-pneumothorax, if the patient be shaken (*Hippocratic succussion*) while the physician's ear is applied to the chest, a *splashing* sound will be heard, which proceeds from the air and liquid in the pleural cavity.

DISEASES OF THE NASAL PASSAGES

ACUTE RHINITIS

(*Acute Nasal Catarrh, Coryza*)

This trouble, familiarly known as a "cold in the head," is a catarrhal inflammation of the mucous membrane of the nose, which often involves also the conjunctivæ, frontal sinuses, pharynx, and Eustachian tubes, and may spread to the larynx, trachea, and bronchi. It is most commonly the result of exposure to cold, either by sitting in a draught, staying out late at night, or by getting wet and failing to change the damp clothes; but it often runs through a household in a way which demonstrates its infective nature. The micro-organisms most frequently found are *Micrococcus catarrhalis, M. paratetragenus, Bacillus septus* (*B. coryzæ segmentosus* of Cautley), *B. influenzæ,* and Friedländer's bacillus. According to Allen, acute and subacute tracheal catarrhs are nearly always due to the first two organisms, acute faucial and nasal catarrhs to the first of these and to Bacillus septus, and chronic nasal catarrhs to Friedländer's bacillus. They may, of course, be associated together, and there may be secondary infection by streptococci, staphylococci, and pneumococci.

An attack of sneezing or a raw or rough feeling in the throat may be one of the first symptoms: but these may be preceded by a feeling of indisposition, with chilliness, headache, dryness and soreness of the throat, and loss of appetite. The sneezing is soon followed by the discharge of clear mucus from the nose; and there is a feeling of stuffiness in the nose, due to swelling of the mucous membrane. At the same time, the eyes are suffused and water freely, there is pain over the eyebrow from implication of the frontal sinus, the throat is sore, taste and smell are impaired, and there may be deafness from closure of the Eustachian tube. Some febrile reaction is present at the same time. If the catarrh extends to the larynx, the voice is hoarse, and there is constant irritating

cough; and its further spread to the lungs will cause the symptoms described below under Bronchitis. Often after two days the acute stage subsides, and recovery seems to be attained: when the discharge again appears, gradually becomes thicker and more opaque, and ultimately quite purulent. It may continue thus for a variable period, from two or three days to two or three weeks. During this time the patient is liable to fresh exacerbations of the inflammation.

Treatment.—In mild forms little requires to be done. Free sweating at night, by means of additional blankets, a hot bath, or diaphoretic drugs, often seems to check the disease; if cough be troublesome, a few drops of ipecacuanha wine, with spirits of nitrous ether, or compound tincture of camphor, will relieve. Local remedies are sometimes useful; for instance, a mixture of bismuth. subnit. ʒvj., morph. hydrochl. 2 gr., pulv. acaciæ ʒij.; 2 or 3 drachms to be snuffed up in small quantities, in the course of a day (Ferrier); or menthol, 1 part, ammon. chlor., 3 parts, acid. boric., 2 parts; or the fluid extract of hamamelis may be snuffed up from the hand (Osler); or steam may be inhaled, impregnated with eucalyptus oil by placing five or six drops of the oil in boiling water in a suitable inhaler; or the nose may be irrigated with a solution of tincture of belladonna, ʒj. in water ℥j.; or a solution of cocaine (2 to 4 per cent.) or adrenalin chloride (1 in 5000) may be sprayed into the nostrils. Internally, Allen recommends oil of cinnamon in doses of 15 or 20 minims in milk, at first hourly for three hours, then two doses of 15 minims at intervals of two hours, then 10 minim doses every three hours; or eucalyptus oil in 10 to 15 minim doses upon sugar every three or four hours.

The treatment by vaccines, after determination of the specific organism concerned and of the opsonic reactions, has also had some success; and for extremely susceptible persons the prophylactic use of vaccines at intervals is recommended.

It will be remembered that acute rhinitis occurs as a specific lesion in some of the infective diseases, such as influenza, measles, diphtheria, congenital syphilis, glanders, and others.

CHRONIC RHINITIS

This is seen in two forms. In the one, *chronic hypertrophic rhinitis*, the mucous membrane of the nose and the lower turbinated processes is greatly thickened, and this thickening may extend to the pharynx and involve the orifices of the Eustachian tubes. It is sometimes the result of prolonged acute rhinitis, at others it is caused by constant mechanical irritation. The breathing is much obstructed, and takes place chiefly by the mouth; and the sense of smell is impaired.

Chronic atrophic rhinitis, in which the mucous membrane is atrophied, is one of the causes of the offensive purulent discharge known as *ozœna.* The mucous membrane is thinned and crusts

collect on the surface, which may be abraded, but is seldom ulcerated. The sense of smell is lost.

Treatment.—For the hypertrophic form, frequent sprays or douches of antiseptic solutions, containing carbolic acid, boric acid, borax, must be used; and if there is much thickening it may be lessened by the use of the galvano-cautery. The hygienic surroundings of the patient also require attention. For the atrophic form, the treatment is very similar but less promising: crusts must be removed and antiseptics applied directly or by douche or spray. Tonics, such as iron, or arsenic, or cod-liver oil, are helpful.

HAY FEVER

(*Summer Catarrh. Hay Asthma.*)

This is a very severe catarrh which occurs in certain individuals, year after year, in the early part of the summer—that is, during June or July, when grasses and other plants are flowering. The symptoms may be chiefly nasal, like those just described, or chiefly bronchial, when the disorder may be called hay asthma. It is undoubtedly, for the most part, due to the diffusion of pollen-grains in the air, and their contact with the nasal, conjunctival, or bronchial mucous membrane, in persons peculiarly susceptible to this form of irritation. Such persons are more often men than women, are generally among the middle and higher classes of society, of neurotic disposition, and have their first attack before the middle period of life. Sometimes a chronic hypertrophic rhinitis is present, rendering the individual constantly liable to catarrhal attacks; and this, or a like morbid condition, is thought by some to be constant and primary. By others the disease is regarded as a part of pulmonary asthma.

Treatment.—Residence in the country during the hay time and exposure to the emanations of grasses must be avoided; if the sufferer goes out, he may wear a veil over the eyes or nose. Locally, M. Mackenzie recommended a spray of a 4 or 6 per cent. solution of cocaine to the eyes; for the nose, the cocaine spray should be followed by the daily introduction along the floor of the nose of a bougie, smeared with vaseline or oil, and left in for ten minutes at first, and for gradually increasing periods up to half an hour or longer. Adrenalin chloride may also be used, either sprayed into nose or throat from a solution of 1 to 5000; or injected hypodermically in a dose of 5 minims of a solution of 1 in 1000. Where there is chronic hypertrophic rhinitis, the application of the galvano-cautery to the swollen mucous membrane, after the preliminary use of a 2 per cent. solution of cocaine, seems to be quickly curative. Internally, valerianate of zinc and asafœtida, quinine, arsenic, belladonna, and bromide of potassium, are of value. If the symptoms are chiefly bronchial, they may be treated, like ordinary asthma, by burning nitre-paper or by smoking stramonium cigarettes.

EPISTAXIS

Epistaxis, or bleeding from the nose, may depend upon local or general conditions. Among the former are acute catarrhal, and tubercular and syphilitic lesions; and traumatic lesions, such as blows and picking. Epistaxis is not uncommon in childhood and early youth from causes that are not always obvious, unless it be simply delicacy of tissue; it is less common in middle age, but is again frequent in elderly people, whose vessels are beginning to degenerate. It is thus related to atheroma. It occurs also at all ages in connection with Bright's disease, cirrhosis of the liver, cardiac valvular disease, the various diseases of the blood, such as scurvy, purpura, and different forms of anæmia, and some infectious diseases, such as enteric and relapsing fevers.

The bleeding is often from the anterior part of the septum; if blood flows from the posterior nares it may trickle down the fauces into the stomach, and be subsequently vomited or passed *per rectum;* or it may cause cough and give rise to a suspicion of hæmoptysis. A pre-existing headache is sometimes relieved by a moderate bleeding.

Epistaxis, even if abundant, usually ceases of itself, but it may recur so frequently, and thus cause so much anæmia, that treatment becomes essential. It may be stopped by keeping the patient in a sitting posture, raising the arms above the head, and applying an ice-bag to the back of the neck. If this is not sufficient the local application (by means of a plug of cotton wool) of a solution of suprarenal extract, or of adrenalin chloride solution (1 in 5000) should be tried. Failing this, the anterior or posterior nares should be plugged, and ergot, calcium chloride, or other hæmostatic may be given internally, or ergotin subcutaneously.

DISEASES OF THE LARYNX

LARYNGITIS

Laryngitis, or inflammation of the larynx, may be acute or chronic, and arises from a number of causes. Amongst these are—exposure to wet and cold, and the ordinary conditions of catarrhal inflammation; contact with irritating vapours, air charged with dust or other minute particles, or scalding water; the impaction of foreign bodies, or direct injury in other ways; extension of inflammation from surrounding parts, the pharynx, the bronchi and trachea, or the tissues outside; the growth of tubercle, cancer, and syphilitic gummata; the circulation of poisons in the blood, such as those of diphtheria and measles, and finally Bright's disease. The results differ some-

what according to the cause, and one can readily distinguish a catarrhal laryngitis, an œdematous laryngitis, the membranous laryngitis which is characteristic of diphtheria, and the laryngitis of phthisis and of syphilis.

ACUTE CATARRHAL LARYNGITIS

Ætiology.—This is mostly the result of exposure to cold air, but also arises from irritating vapours, dusty air, the entrance of foreign bodies, and inflammation spreading from the posterior nares, pharynx or bronchi. It is one of the effects of the poison of measles, and less frequently of other infections.

Morbid Anatomy.—The disease consists of swelling and increased vascularity of the mucous membrane of the larynx, with the secretion of more or less mucus, or in later stages muco-pus. Occasionally slight abrasions of the epithelium occur, and less frequently hæmorrhage takes place into the tissue of the mucous membrane or on the surface. In very severe cases there is œdema of the submucous tissues. As a result of the inflammation of the overlying structures certain changes take place in the neuro-muscular apparatus of the larynx, especially paralysis of the thyro-arytenoid muscles (internal tensors).

Symptoms.—There is at first soreness or dryness of the throat, and the voice becomes hoarse or entirely lost. There is occasional irritating cough, shrill, husky, or toneless, with expectoration from time to time of small plugs of mucus. Respiration is generally but little affected, but there may, in exceptional cases, be some stridor, or mucous râles, produced in the larynx; and in children dyspnœa is much more often a marked symptom. Fever may be slight or none. On examination with the laryngoscope the larynx is seen to be reddened, either generally or in patches, the posterior ends of the vocal cords, the inter-arytenoid space, and the ventricular bands being most frequently affected. In consequence of the thyro-arytenoid paralysis, the cords fail to meet in the middle, leaving a fusiform opening, and to this, as well as to swelling of the inter-arytenoid fold, the loss of voice is to be attributed.

Children are liable to a form of acute laryngitis (*laryngitis stridulosa*), which is characterised by the sudden development of suffocative symptoms, frequently in the middle of the night. During the day there is only slight cough and huskiness, but some time in the night the child wakes up suddenly in terror, with severe dyspnœa, and a barking or husky cough, followed by loud and prolonged crowing inspiration. The voice is husky and feeble, and the features are congested; if the condition continues, the face may become pale and livid, and suffocation seems imminent. Usually, however, in a short time the symptoms become less severe, and the child falls asleep. Either on the same night, after a few hours' sleep, or on subsequent nights, the same attacks of threatening suffocation with croupy inspiration may take place. In association with these

attacks there is more fever (white tongue, flushed face, hot skin, &c.) than commonly occurs in catarrhal laryngitis of adults. The spasmodic attacks are probably due to the inspiration of mucus, which blocks the narrow glottis during sleep. These symptoms are apt to recur in the same child whenever it catches cold; they are, however, rarely fatal.

The **Prognosis** of acute laryngitis is mostly favourable; it generally subsides in the course of a few days.

The **Diagnosis** is generally simple, especially in adults: diphtheria is more severe, and may be accompanied by membrane on the fauces, by the expectoration of membrane, or by albuminuria.

Treatment.—For acute laryngitis the patient should be placed in a uniformly warm atmosphere, and use steam inhalations from a suitable inhaler frequently. This may be charged with tr. benzoin co. (℥j. to a pint of water), or benzoic acid, or ol. pini sylvestris (5 minims with 10 grains of magnesii carb. levis suspended in water ℥j.), or lupulin (℥ss). Sprays of menthol (20 gr. to 30 gr. in liquid paraffin ℥j.), oil of eucalyptus, and creasote are also useful. Demulcent liquids should be drunk freely, or small pieces of ice may be sucked. The irritation of cough should be allayed by opiates. The diet or regimen usual in febrile affections will of course be followed. The patient should abstain as much as possible from using the voice. Local applications by the laryngeal brush seem not to be advisable till the later stages, when astringent solutions may be used like but weaker than those given on p. 464.

For laryngitis stridulosa an emetic is often useful, such as sulphate of zinc (5 to 10 grains), or ipecacuanha (2 to 5 grains of powder, or a drachm of the wine every ten minutes, till vomiting is produced). In addition, hot flannels or a hot sponge should be applied to the throat. In the intervals the laryngitis is to be treated by a warm moist atmosphere (steam-kettle) and small doses of bromides and chloral.

ŒDEMATOUS LARYNGITIS

Ætiology.—This may be a result of laryngitis arising in various ways. It sometimes occurs in catarrhal cases, after the administration of potassium iodide, and in the course of Bright's disease. It is, however, frequently the result of *sepsis*, whether local or general: it thus occurs among hospital nurses, students, and others exposed to such influences; and is often set up by inflammations of the pharynx, diphtheria, disease of the cartilages and perichondrium, such as occur in enteric fever, syphilis, and phthisis, cellulitis of the neck spreading from aneurysm and tumour in the chest and that known as *angina Ludovici* (*see* Diseases of the Mouth). Injury and the contact of boiling water will also cause it. The localised œdema described as angeio-neurotic, frequently occurs in the laryngeal tissues, and is often fatal (*see* Angeio-neurotic Œdema).

Morbid Anatomy.—It consists of an effusion of inflammatory serum into the submucous tissue, and the serum contains many

leucocytes, so that it may be sero-purulent; or actual pus is diffused through the tissue.

Symptoms.—These are often very rapidly developed, and within a few hours the patient may be in imminent danger from the obstruction to respiration produced by the swelling. Besides the dyspnœa, which is the prominent symptom, dysphagia is also experienced, the voice becomes hoarse and feeble, or disappears entirely, and there is some stridor in respiration. The laryngoscope reveals enormous swelling of the epiglottis, which forms a thick semicircular fold, or shows its two lateral halves much swollen and pressed together; in colour it is bright red. The swelling also affects the ary-epiglottic folds and the ventricular bands, but rarely the vocal cords themselves. The swollen epiglottis can be also felt by the finger, or even seen on depressing the tongue with a spatula. Care must be taken not to set up a spasm of the larynx.

The **Diagnosis** is confirmed by the use of the laryngoscope; in its absence the swollen epiglottis may be felt by the finger.

The **Prognosis** in cases of extensive œdema is a serious one: in those which arise from blood-poisoning the latter may be fatal even if the dyspnœa is relieved.

Treatment.—The object should be to remove the laryngeal obstruction as soon as possible. Bleeding by means of leeches to the neck, adrenalin spray (1 in 5000), ice sucked, or ice applied to the neck, potassium bromide in 10 or 20 grain doses, and the subcutaneous injection of pilocarpin nitrate may cause the œdema to subside; but if the dyspnœa is urgent, the swollen epiglottis should be scarified by the laryngeal lancet, or by a curved bistoury, covered nearly to the point with adhesive plaster, and the throat should be gargled with warm water after the operation. If this fails, intubation or a low tracheotomy must be performed. Quinine and iron should be given internally.

MEMBRANOUS LARYNGITIS

Ætiology.—The most common cause of membranous laryngitis is diphtheria, which either begins in the fauces and spreads to the larynx (*see* p. 121), or attacks the larynx at first, without either then, or later, involving the fauces. It is of interest to note that these primary laryngeal cases are more common in children than in adults, and that they are less often accompanied by albuminuria, or followed by paralysis, than those in which the throat first suffers.

A membranous laryngitis also occurs occasionally in connection with other specific fevers, such as scarlatina or measles; and it may certainly be produced by traumatic causes or local irritants, such as chemical vapours, boiling water, or impacted foreign bodies.

Symptoms.—The local symptoms will be like those already described under diphtheria; but the toxic symptoms of an infectious

disease will be absent or little pronounced, when the cause is more distinctly traumatic.

Diagnosis.—In the majority of children taken with dyspnœa, ringing or "croupy" cough, and retraction of the chest, without apparent cause, and threatened with suffocation in from one to four days, membranous laryngitis is present, and in the majority of these again diphtheria is the cause; but it is generally impossible to examine with the laryngoscope, and the first proof of the presence of membrane may be provided at or after the operation for tracheotomy. It is distinguished from laryngitis stridulosa (*see* p. 460) by the more gradual development and more uniform progress of the dyspnœa.

Treatment.—Membranous laryngitis may be treated as shown under Diphtheria, both when it is due to this disease and when it is associated with scarlet fever, measles, or other infectious illness. The antitoxin of diphtheria should be used in the first case.

CHRONIC CATARRHAL LARYNGITIS

Ætiology.—This often follows upon acute laryngitis, especially when the latter is not properly treated with complete rest of voice. It results, also, from the failure to breathe properly through the nose, when the nasal passages are obstructed by deviation of the septum, turbinal thickenings, hypertrophic rhinitis, or adenoids; and is often due to a faulty use of the larynx in those who sing and speak in public. It is also seen in others who use the voice continuously for long periods, and with much exertion, like costermongers, schoolmasters, and clergymen. It may be associated with the pharyngitis, so often induced by the excessive use of alcohol or tobacco; or infection may spread from the mouth in cases of oral sepsis.

Symptoms.—There is hoarseness of voice, accompanied by dryness, irritation of the throat, and tickling on prolonged use of the larynx; or there may be loss of voice. The cough is frequent, but there may be only a little hawking-up; and the expectoration, which is mostly viscid mucus, is never abundant. The symptoms are often most marked after an interval of rest, and disappear during the effort of talking, until after a time fatigue again ensues. Dyspnœa is generally absent. With the laryngoscope, more or less congestion of the mucous membrane is seen; this may be diffused or unequally distributed, and mucus is here and there adherent to the surface. In old cases the mucous and submucous tissues become swollen and thickened, especially over the epiglottis, inter-arytenoid fold, and ventricular bands, and the vocal cords may become thickened, granular, or have nodules upon them (*chronic hypertrophic laryngitis*). The mobility of the vocal cords is impaired, partly by a paralysis of some of the muscles of the larynx, which, according to Ziemssen, is more often unilateral than bilateral; partly by the thickening of the mucous membrane. Indeed, the swollen inter-arytenoid fold may project between the cords on

phonation so as to hinder their closure. Erosions, or shallow ulcers, are often present, especially on the cartilaginous part of the vocal cords, and between the arytenoid cartilages.

Diagnosis.—This must be made from the history and the laryngoscopic appearances, in which the thickening has to be distinguished from the transparent swelling of œdema and the dull red swellings of tubercular laryngitis. In cases of long standing a possible connection with phthisis must be carefully considered; in older patients cancer may cause a chronic thickening, which is likely to affect one cord only, and to impair its movement.

Treatment.—The condition is very troublesome, and requires persistent treatment. The patient should protect himself from cold and exposure by suitable clothing, as well as by confinement to the house in bad weather; and he should talk as little as possible. Attention to the bowels, and the use of iron tonics and strychnine are desirable; and change of residence to a mild and equable climate may be beneficial, or even necessary. Astringents should be applied by means of the laryngeal brush. For this purpose the following solutions may be used: Iron perchloride, 10 to 20 grains to the ounce of water; copper sulphate, 10 grains; zinc chloride, 30 grains; silver nitrate, 10 to 30, or even 60 grains; zinc sulphate, 10 grains; alum, 30 grains. One of these should be employed daily for seven days, on alternate days during the next two weeks, and so on with gradually decreasing frequency. Similar solutions may be sprayed upon the larynx by a suitable apparatus, two or three times daily, beginning with weaker solutions and gradually increasing their strength. For cases with excessive secretion turpentine may be locally applied; and carbolic acid (℥ss–℥j. to glycerin ℥j.) for cases with long-standing hyperæmia and diminished secretion. The paralysis of chronic laryngitis should be treated by electricity applied internally.

In *subglottic chronic laryngitis* the mucous membrane below the vocal cord becomes much thickened and hypertrophied, so as to be easily visible like a second true cord. It is sometimes a result of disease of the cricoid and arytenoid cartilages. The prognosis is very unfavourable, and active treatment is required of the same kind as that employed in ordinary chronic laryngitis to prevent hypertrophy. In late stages bougies may be required to dilate the glottis, and tracheotomy has been several times found necessary.

Glandular laryngitis, in which the racemose glands are chiefly affected, is generally associated with follicular laryngitis. The symptoms are those of a mild chronic laryngitis, and require astringent treatment. Nitrate of silver solution, 20 grains to an ounce of water, has been especially recommended.

Laryngitis sicca and *atrophic laryngitis* occur in association with atrophic rhinitis and present similar conditions, the formation of crusts on a thinned and abraded mucous membrane. *Pachydermia laryngis* is a chronic fleshy thickening occupying mostly the posterior thirds of the cords, and *singers' nodes* are localised thickenings on the

free edge and upper surface of the cords, generally at the junction of the anterior and middle thirds.

The last two require the stronger astringent applications: salicylic acid in alcohol (1 to 8 per cent.) may be applied locally. For singer's nodes prolonged rest, local treatment of the nose and pharynx, and later suitable vocal exercises are also desirable. Exceptionally, they may be removed by operation or the galvano-cautery.

LARYNGEAL PERICHONDRITIS

This is mostly the result of phthisis, carcinoma, syphilis, or enteric fever affecting the larynx, or of simple chronic laryngitis. It may also arise from traumatic causes, such as direct injury by a blow or from cut throat, an impacted foreign body, the pressure of the larynx against the spinal column in patients confined to the recumbent position, or the frequent passage of bougies down the œsophagus. As a result of the inflammation the perichondrium becomes thickened, pus forms in its fibres and collects between it and the cartilage, which, separated from its nutritive supply, becomes necrosed. The structures superficial to the perichondrium also become inflamed, œdematous, and purulent; thus an abscess is formed, which contains the dead fragment of cartilage. This sequestrum may remain for months or years after the opening of the abscess; on its removal the parts will contract and cause deformity and stenosis of the larynx.

Symptoms.—Dull, aching pain, tenderness on manipulation with difficulty of swallowing, and hoarseness of voice or aphonia, are the usual symptoms, varying somewhat according to the cartilage affected, and often very much masked in secondary cases; thus in typhoid fever, loss of voice may be the only symptom suggesting an inquiry into the condition of the larynx. If the membrane on the outer side of the thyroid is affected, there will be swelling in the neck and formation of an abscess. If the inner surface of the thyroid, or the cricoid or arytenoid cartilage is affected, there is œdematous infiltration of the interior of the larynx corresponding to it, which will be visible with the laryngoscope. The mobility of the vocal cords may be lessened by paralysis of the posterior crico-arytenoids when the cricoid cartilage is diseased, and by direct implication of the cord when one arytenoid is affected.

The **Prognosis** is unfavourable; when the primary cause, as typhoid or syphilis, is not in itself fatal, troublesome contractions of the glottis ensue after discharge of the cartilage; and pneumonia and gangrene of the lung have occurred from insufflation of the septic secretions.

Treatment.—In acute cases, the inflammation may be reduced by the application of leeches or an ice-bag to the surface. If an abscess forms it must be incised—from within if the arytenoid or epiglottis, by incision in the neck if the thyroid or cricoid cartilage,

is concerned. Often, however, tracheotomy is required, and before the tube can be dispensed with the contraction of the glottis has to be treated perseveringly by dilatation or other operative measures.

TUBERCLE OF THE LARYNX

It is well known that amongst patients suffering from phthisis a considerable number have a laryngeal affection, which has been described as *laryngeal phthisis*. This is due to the actual deposit of tubercle in the laryngeal tissues; and it is nearly always secondary to the formation of tubercle in the lungs. The tubercles occur as minute collections of cells in the mucous or submucous tissues, forming, perhaps, slight prominences on the surface, leading in time to more or less, often considerable, œdema of the surrounding parts, and later to ulceration. Extending more deeply in severe cases, with the assistance of pyogenic organisms, the inflammatory process leads to deep ulceration, to perichondritis and to necrosis of the cartilages. The most frequent seats of the deposit are the mucous membrane covering the epiglottis and the arytenod cartilages, the ary-epiglottic folds, the ventricular bands, and the vocal cords. Paralysis of the vocal cords may be simultaneously caused by thickening of the right pleura involving the right recurrent laryngeal nerve, or tuberculous bronchial glands pressing upon one or both.

The **Symptoms** are those of a chronic laryngitis, and in cases of ordinary severity consist of hoarseness of voice, frequent husky cough, and pain on swallowing. Sometimes in early stages the voice may be lost entirely from functional failure, in later stages, from paralysis of a vocal cord, or extensive destruction; and swallowing may be not only painful but difficult on account of swelling of the tissues, or from their destruction preventing perfect closure of the larynx. The cough is occasionally severe and paroxysmal, and expectoration is variable, depending rather on the condition of the lungs than on that of the larynx. In a small number of cases, considerable obstruction to respiration arises. In early stages the laryngoscope shows pallor of the mucous membrane; and a decided anæmia of the larynx occurs quite early in many cases of phthisis. When infiltration takes place the parts often assume a characteristic appearance, the ary-epiglottic folds on one or both sides being swollen up into a pale globular or pyriform tumour—the base backwards, the point forwards; and when both are affected the swellings coalesce in the middle line. The epiglottis often forms a turban-shaped swelling; and the same thickening may affect the ventricular bands, which are, however, often concealed. Subsequently, ulcers form upon the swollen tissues as well as upon the vocal cords, especially in their posterior halves. A frequent characteristic is ulceration with granulations in the interarytenoid space.

Diagnosis.—This must be made partly from the laryngoscopic

appearances, and partly from the condition of the lungs, as in many cases there is obvious phthisis. The pyriform swellings of the ary-epiglottic fold are characteristic of the condition; but when they are absent there may be difficulty in distinguishing it from *chronic catarrhal laryngitis* and from *syphilitic* disease. In the former there are less swelling and more congestion than in tubercular laryngitis; in syphilis the ulcers are generally larger and deeper, situate upon a more inflamed base, and solitary; the thickening is more irregular, and the disease often unilateral. The ulceration of cancer is sometimes difficult to distinguish from that of tubercle; cancer is often unilateral, and occurs in older patients.

Prognosis.—Cure or permanent arrest of the symptoms is rare, but the condition is not often directly fatal.

Treatment.—In early stages relief is obtained by the use of mineral astringents, as in chronic laryngitis. Perchloride and sulphate of iron have been especially recommended. Inhalations of the vapours of compound tincture of benzoin (℥ss. in a jugful of boiling water) or of lupulinum (℥ss.) are of value. Where the cough is troublesome the laryngeal insufflator may be used. This is a tube with a curved nozzle, which is introduced into the back of the mouth over the larynx, with the aid of the laryngoscope, and powders can be blown through it on to the larynx. Morphia acetate ($\frac{1}{8}$ gr. to $\frac{1}{2}$ gr.), with $\frac{1}{2}$ gr. of starch, or one or two grains of a mixture of morphia acetate 1 part, boric acid and iodoform 2 parts each, and starch 7 parts; or orthoform 3 to 5 grains may be thus employed. A cocaine spray (10 per cent.) and menthol pastilles are also useful.

Where there is ulceration some success has attended the repeated use of an aqueous solution of lactic acid (20 to 80 per cent.), which should be applied firmly by means of a pledget of lint; and the surface may in some cases be with advantage scraped by a *curette*, both to remove diseased tissue and to help the action of the acid. Rosenberg advocates the injection of a 20 per cent. solution of menthol in olive oil. Fifteen minims are used at the first sitting, and larger quantities afterwards. Fluids thickened with arrowroot, cornflour, or isinglass are best given where swallowing is difficult from food entering the larynx, or, in extreme cases, an œsophageal tube may be necessary. Œdema may have to be treated with scarification, and tracheotomy may be required in urgent dyspnœa. The general treatment suitable to cases of phthisis must at the same time be continued.

SYPHILIS OF THE LARYNX

Syphilis affects the larynx in many ways: in the hereditary form in infancy and childhood; in the acquired form in secondary and tertiary and intermediate stages. The secondary lesions of acquired syphilis are chronic hyperæmia, superficial ulcerations, and condylomata or mucous patches, of which the last are very rare. In the

later stages of the disease, gummata and deep ulcerations occur. The former are round elevations of the same colour as the rest of the larynx, from the size of a pin's head up to that of a pea, generally single and situate on the posterior wall of the larynx, the epiglottis, ventricular bands, or ary-epiglottic folds. They may ulcerate deeply or become absorbed. The ulcers of late syphilis, though in themselves not unlike tubercular ulcers, solitary, or only two in number, and often confined to one side of the larynx, are generally deeper, and surrounded with more inflammatory redness. They also develop very rapidly in a few days. Laryngeal œdema, and perichondritis with laryngeal necrosis, occasionally result, and the cicatrisation of ulcers frequently leads to the union of parts of the larynx to each other, or to the pharynx, so that serious distortions of the larynx or contractions of the glottis ensue. Thus the cords may be united by a web, or the epiglottis may be fixed to the pharynx.

Symptoms.—These are not distinctive, and vary much according to the severity of the lesion. They are hoarseness or loss of voice, occasionally cough in earlier stages, and more or less dyspnœa in later stages. Swallowing is often painful, though the absence of pain when not swallowing is remarkable. Muco-purulent expectoration with blood may accompany extensive ulceration, and in a few cases free hæmorrhage has taken place.

Diagnosis.—Syphilis of the larynx may be confounded with tubercle and with cancer. In this last the ulcers are generally preceded and accompanied by growth, in the form of nodular excrescences; they may be very large, and the surrounding tissue is inflamed. The diagnosis of syphilitic laryngitis should not be hastily made from the history alone, without a laryngoscopic examination. Dyspnœa and stridor in such persons may arise from paralysis of the vocal cords, from aneurysm pressing on the trachea or laryngeal nerves, or from syphilitic stenosis of the trachea or one bronchus.

Prognosis is not specially unfavourable to life.

Treatment.—Syphilitic lesions yield to the vigorous use of mercury (especially by inunction) or of iodide of potassium. The resulting contractions may necessitate tracheotomy or intubation. The voice is then commonly lost, and generally a tube has to be worn for life; but attempts may be made to dilate the glottis mechanically, or to divide a web by the cutting forceps, or dilator, or by the electric cautery.

LUPUS OF THE LARYNX

Lupus, of which a fuller description is given under Diseases of the Skin, rarely attacks the larynx; although the systematic examination of this part in persons with lupus of the skin has shown it to be often present when not in the least suspected.

It occurs as pale or dark-red nodules, or papillary outgrowths, isolated or grouped upon a hyperæmic base, appearing first and most often upon the epiglottis, then upon the ary-epiglottic folds, and posterior wall of the larynx, and least often upon the vocal cords. As they increase in size they cause a general thickening of the parts. Later on, ulceration takes place, and the ulcers are followed by scars, in which again fresh nodules may appear; but the cicatrisation rarely leads to high degrees of stenosis. Some nodules may be absorbed without ulceration.

Symptoms.—There are some soreness, trouble in swallowing, and alteration in the voice, which becomes hoarse or is even lost. Dyspnœa may occur in late stages.

Treatment.—This has consisted chiefly of local applications of tincture of iodine, iodoform, nitrate of silver and lactic acid, and the use of the electric cautery, or of the curette, as in tubercle (*see* p. 467). The open-air treatment should also be employed, and arsenic and cod-liver oil should be given internally.

TUMOURS OF THE LARYNX

BENIGN TUMOURS

These are papilloma, fibroma, mucous cyst, myxoma, angioma, and lipoma. The last three are quite rare, and occur especially in children.

Papillomata are the most common. They are frequently about the size of a pea, but may be as small as a mustard-seed, or in rare cases as large as a walnut. They are pink, whitish-gray, or red, have an uneven, or papillated, or warty surface, and grow mostly from a broad base. They are often multiple, and their usual seat is the vocal cord on one or both sides, or the angle between the cords; sometimes the ventricular bands or the epiglottis. They are liable to recur after removal, and in a few instances have been known to undergo epitheliomatous degeneration.

Fibromata, or fibrous polypi, are of slower growth, and show no tendency to recur. They are round or oval, sessile, or pedunculated, with a smooth surface and bright red colour. Usually of hard consistence, they are more rarely soft, and contain a good deal of serous fluid in the meshes of the fibrous tissue. They are generally single, and arise from one of the vocal cords.

Mucous cysts arise commonly from the epiglottis, and are surrounded with an area of injected mucous membrane. They have dense walls, and are filled with thick, white, sebaceous material, or thinner yellowish or brown fluid.

The **Symptoms** of tumours depend upon their seat. If situate upon the vocal cords the voice is impaired or lost; and impairment of voice is the commonest symptom. If the tumour is sufficiently large, dyspnœa is present. Dysphagia from implication of the

epiglottis, and cough, generally dry and hacking, also occur. In children the cough may be croupy. Pain appears to be rare.

The **Treatment** is removal by surgical operations, for the details of which the reader is referred to surgical works, or special treatises.

MALIGNANT TUMOURS

These are mostly epithelioma, but scirrhus, encephaloid cancer, and sarcoma also occur. They are more frequent in men than in women, and appear commonly in advanced life. The posterior part of the cricoid cartilage and the vocal cords appear to be the most frequent seats of commencement of the growth (Semon); but it may begin in the epiglottis, or the ventricular bands, and it may involve the whole larynx, so that after a time it is impossible to say where it has begun. The appearance is at first not distinctive; it may be an infiltrating or warty growth, or a definite tumour. But it shortly ulcerates, vegetations spring up about the margins in epithelioma, and these ulcerating in their turn, the disease rapidly spreads. The surface is often covered with pus, or sanguineous muco-pus, and occasionally free hæmorrhage takes place. Œdematous laryngitis and perichondritis occur as complications. The larynx is, of course, affected sometimes by cancer spreading from the pharynx or the neck.

Symptoms.—Dyspnœa, dysphagia, and alteration of the voice occur as in other laryngeal affections. An important feature is *pain*, which is at first local, but subsequently radiates to the ear, orbit, or forehead. As ulceration proceeds, the breath becomes fœtid, and hæmorrhage may occur. The voice early becomes hoarse, but is rarely completely lost. Occasionally the submaxillary glands are implicated, and in rare cases the thyroid cartilage is distended by the growth within.

The **Diagnosis** is often difficult at first, but on the appearance of a tumour its features, the age of the patient, and the absence of a history of syphilis, or of long-continued chronic laryngitis, will afford strong evidence of its cancerous nature.

The **Prognosis** is unfavourable, and the only successful **Treatment** must be surgical. A sufficient number of operations for the removal of the growth have been successful to justify this procedure being considered whenever an early diagnosis can be made.

FOREIGN BODIES IN THE LARYNX

There are a large number of foreign bodies which have at different times found their way into the larynx. Among these are peas, beans, buttons, coins, fragments of bone, shells, pebbles, artificial teeth, portions of solid food, and pieces of children's toys. The obstruction may be at once fatal, or it may be so slight as to be scarcely noticeable; indeed, it is not always known to the patient

or to his friends that a foreign body has been introduced. Thus a child was suddenly taken with "convulsions," became black in the face, and died. It was only on *post-mortem* examination that it was discovered that a pea had fallen into the larynx, where it lay completely occluding the glottis. A man came to the hospital for hoarseness and occasional cough. On examining the larynx I saw a piece of bone below the vocal cords. When told this, he remembered that *three months* previously he had by accident swallowed a piece of bone in some sheep's head broth, that it stuck in his throat and nearly choked him, but that his wife forced a passage with the gravy-spoon. He was relieved, and, though the laryngeal trouble followed, he still supposed that he had properly swallowed the bone. Mr. Golding-Bird removed it by laryngotomy, and it measured three-quarters by five-eighths of an inch.

Impacted foreign bodies may give rise to secondary inflammation, œdema, or hæmorrhage, or to pain and spasm. Sometimes a change in their position may cause sudden death. Their position and relations should be accurately estimated, and they should be removed, if possible, by forceps through the upper opening of the glottis, even when they are below the cords. But they may require thyrotomy, or infra-thyroid laryngotomy.

PARALYSIS OF THE LARYNGEAL MUSCLES

From the peculiar course of the recurrent laryngeal nerve—the chief motor nerve of the larynx—paralysis of these muscles has often a diagnostic importance beyond that of the trouble arising locally. But it may be caused by lesions not only of the laryngeal nerves, but also of the vagus above their origin, and of the medulla oblongata where the nuclei are situate. Thus laryngeal paralysis is a part of bulbar paralysis, results from syphilis and tumours affecting the medulla oblongata, and occurs occasionally in association with locomotor ataxy, general paralysis, syringomyelia, and insular sclerosis. The vagus in the neck may be compressed by tumours and enlarged glands, or may be injured by wounds or operations. The recurrent laryngeal nerves are in danger in two situations—the thorax and the neck; and the left is the more liable to lesion from its curving round the arch of the aorta, whereas the right goes no lower than the subclavian artery. Either of them may be involved in the fibrous thickening at the apex of the lung in chronic phthisis, but the left is especially liable to be compressed by aneurysm of the arch of the aorta, by mediastinal tumours, and by enlarged bronchial glands. In the neck the two nerves ascending to the larynx lie between the trachea and the œsophagus, and may be involved together in cancer of the latter, or compresssed by an enlarged thyroid body. Paralysis also occurs, as already stated, in catarrh, and from other local affections of the larynx; as a result of diphtheria, influenza, and other infectious

diseases, chronic alcoholism, and poisoning by lead and arsenic; and finally, as a purely functional failure, or part of hysteria.

COMPLETE PARALYSIS OF THE VOCAL CORDS

This is the result of lesions dividing or severely injuring the recurrent laryngeal nerve, or the vagus above; or of diseases of the medulla oblongata—*e.g.*, bulbar paralysis. Of the nerve-lesions, neuritis from alcohol and diphtheria must not be forgotten. The paralysis may be bilateral, which is very rare, or unilateral. When bilateral, the cords assume what is known as the *cadaveric position.* They are immovably fixed midway between the positions of *ab*duction and *ad*duction, each cord with a concave margin, leaving a fusiform interval between them, unaffected by inspiration, expiration, or attempts at phonation. The aperture is sufficiently wide for respiration, hence there is no dyspnœa; but phonation is impossible, as the cords cannot be approximated, so that the patient speaks in a whisper, and the acts of coughing and expectoration are imperfectly performed. Some stridor is produced on forced inspiration, probably from the vocal cords, ary-epiglottic folds, and arytenoid cartilages being thrown into vibration.

When the paralysis is unilateral, the affected cord assumes the cadaveric position, while the sound cord has its full mobility. Again, there is no dyspnœa. The voice may be entirely lost, but often it is produced by the arytenoid of the healthy side being drawn completely over the median line till it comes into contact with the paralysed cord; so that the glottis lies obliquely with its anterior end in the middle line and its posterior end much to the affected side. The voice is then harsh sometimes, and may break into falsetto on an increase of the effort. The cough has often a peculiar brassy or clanging quality.

PARALYSIS OF THE ABDUCTORS

Although the recurrent laryngeal nerves, supplying as they do all the muscles of the larynx except the crico-thyroid, must contain fibres for both *ad*ductors and *ab*ductors, it is a remarkable fact that coarse progressive lesions of these nerves (compression by tumours or aneurysms) result at first in paralysis of the *abductors* alone; later only are the internal tensors (*thyro-arytænoidei*) affected, and last of all the chief adductors (*crico-arytænoidei laterales*). The abductor fibres form a separate bundle lying internal to the adductor fibres in the recurrent laryngeal nerve of the dog (Risien Russell); but their greater liability to suffer from lesions affecting the whole nerve or centres is apparently due, as shown experimentally, to less powers of resistance to external influences. Abductor paralysis also results from lesions of the medulla, where it may be supposed it sometimes depends on a separate affection of the nucleus of the abductor fibres; though it is to be noted that adductor paralysis alone never arises

under such circumstances. Pressure on the trunk of one vagus nerve may cause double abductor paralysis by reflex influence. Abductor paralysis is probably sometimes the result of a change primarily in the muscle; it is rarely the result of hysteria.

The first effect of the lesion is that the cord during respiration not being fully abducted remains in the cadaveric position, and allows ample space for the passage of air; after a time, however, the antagonistic muscle, or adductor, contracts (*paralytic contracture*), and the cord is drawn into a position of adduction. Thus, in bilateral paralysis of the abductors the cords are seen to be permanently approximated in the middle line to within one-tenth of an inch of each other; on attempted phonation they meet completely in the middle line; on inspiration they do not separate, but are even drawn a little closer together; on expiration they scarcely move, or only in the reverse sense to their slight movement in inspiration. The important symptom is *dyspnœa*, which results from the permanent narrowing of the glottis; this is generally accompanied by *stridor* on inspiration, which is worse on exertion, and often extremely loud during sleep. The voice is clear, or it may be a little hoarse. Coughing can be perfectly effected.

When only one cord is paralysed, dyspnœa only occurs on exertion, and the stridor is less or absent. On phonation the healthy cord meets the paralysed cord in the middle line; and the voice is normal.

Diagnosis.—Abductor paralysis may be confounded with spasm of the abductors, with ankylosis of the arytenoids in the position of adduction, and with perverted action of the cords in which they move inwards instead of outwards during inspiration. It is important to remember that the lesion which causes unilateral or bilateral abductor paralysis may at the same time cause narrowing of the trachea, by pressure (aneurysm, tumour), or by cicatrix (syphilis); and the dyspnœa and stridor due to the latter may be wrongly attributed to the former. Tracheal stenosis usually causes expiratory as well as inspiratory stridor.

The **Prognosis** is generally serious. Except when hysteria or syphilis is the cause, there is little hope of recovery; and while some cases may last for years without any change, there is a constant liability to death from suffocation. If the adductors are subsequently paralysed the obstruction to breathing is diminished but aphonia ensues. Death may arise from the primary lesion, such as œsophageal cancer, or double aneurysm. In long-standing cases the posterior crico-arytenoid muscles become completely atrophied.

Treatment.—If the cause of double paralysis is central, or if there is a possibility of syphilis being the cause, iodide of potassium and perchloride of mercury may be given internally, and the tone of the muscles improved by faradism and galvanism applied with laryngeal electrodes; the subcutaneous injection of sulphate of strychnia ($\frac{1}{30}$ gr.) has also been recommended. But if no improvement takes place in a few weeks, and if dyspnœa is constant, or

night attacks take place, tracheotomy should be performed, and the tube worn constantly.

In unilateral paralysis the risk of asphyxia is much less, and the treatment may be directed mainly to the cause.

PARALYSIS OF THE ADDUCTORS

This is mostly the result of functional disease or hysteria, and rarely occurs alone from structural lesions. The adductors are the lateral adductor, or *crico-arytænoidei laterales*, and the central adductor, or *arytænoideus proprius;* the inner fibres of the *thyro-arytænoidei*, or internal tensors, also act as adductors of the anterior portions of the vocal cords. In the most common form of adductor paralysis these are all affected. When examined with the laryngoscope, the glottis is seen to be widely open: on attempts to speak the cords scarcely move, but remain still at the sides of the larynx. As the cords cannot be approximated, the patient speaks only in a whisper, no laryngeal voice being produced, though sometimes, with an effort, a momentary contact of the cords may be effected. Coughing, in which the cords are brought together by involuntary reflex action, is generally perfect; and from the open condition of the glottis there is no dyspnœa. This constitutes *functional* or *hysterical aphonia*, which is, however, often started by slight catarrh of the larynx, or by sore throat, or by other local trouble, both in definitely hysterical persons and in others suffering from anæmia or general weakness.

Sometimes the adductor paralysis is less extensive; the internal tensors may be alone affected, so as to produce want of contact of the cords on attempted phonation, each cord presenting in its anterior half a concave margin towards the middle line. And sometimes the central adductor is paralysed, in which case the anterior portions of the cords come into contact, and a triangular space is left open behind, between the arytenoid cartilages. These last two forms are not uncommon in the course of catarrhal laryngitis. They may occur together, producing defective closure in front and behind, while the processus vocales are in contact. In these cases the loss of voice is not so complete as in that first described.

The **Diagnosis** of these conditions is easily made with the laryngoscope. Even without this, the voicelessness of the patient, the absence of dyspnœa, cough, and expectoration, and the power to cough at will, are sufficiently distinctive.

The **Prognosis** is favourable, and cases of many years' duration may be at length cured.

Treatment.—Functional aphonia must be treated by faradisation. Slight cases will often be cured by the application of a current to the neck, an electrode being placed on either side of the larynx. But long-standing cases require endo-laryngeal faradisation. One electrode should consist of a metal plate placed on the neck in front of the larynx, and retained by a band round the neck.

The other is the laryngeal electrode, which must be introduced into the larynx with the aid of the mirror. The instrument is so made that the current passes to the free extremity (touching the larynx) only when a key in the handle is depressed by the operator. It is advisable in all cases to give a powerful shock even at first. Often the patient at once cries out, and recovers the use of the voice from that time. In other cases the voice is recovered for twenty-four hours or more, and is lost again till the muscles are again submitted to faradisation. Stimulating applications, inhalations, and sprays are also useful as adjuncts. The general health of the patient should be considered, and nervous failings should be met by the requisite moral treatment. Paralyses occurring in the course of laryngitis will generally spontaneously recover with the causative lesion.

PARALYSIS OF THE EXTERNAL TENSORS

The crico-thyroid muscles may be paralysed on one or both sides, but this is not a common occurrence. It arises from cold, or from prolonged use of the voice. The voice becomes gruff, or may be entirely lost. With the laryngoscope, the cords are seen to be applied to each other in a wavy line, instead of being perfectly straight and parallel. This condition should arise from lesions of the superior laryngeal nerve, such as the pressure of an inflamed gland; but it is more often seen as part of or in connection with diphtherial paralysis.

Paralysis of the thyro-epiglottic and ary-epiglottic muscles, which depress the epiglottis, occurs also from lesion of the superior laryngeal nerve, and is accompanied by anæsthesia.

SPASM OF THE GLOTTIS

In this affection the adductors are spasmodically contracted, and complete closure of the glottis takes place, preventing the entrance of air, and producing asphyxia, or even death. It may occur at all ages, but is especially frequent in infants, in the form now to be described.

LARYNGISMUS STRIDULUS

(*Spasmodic Croup. Child-croning*)

This occurs between the ages of three months and two years, and is more common in boys than in girls. It is promoted by imperfect hygienic conditions, and is more frequent among the poor, and in those children that are hand-fed, or nursed by sickly and half-starved mothers. Rickets, which occurs under similar hygienic conditions, is observed in a large proportion (75 per cent.) of the cases. In those liable to it, a number of causes may excite a spasm, such as crying, sucking, quick movements, milk getting down the

larynx, indigestible food in the stomach, the irritation of dentition, and the presence of post-nasal adenoids; but the attacks often occur without any such obvious antecedent. The child may be in fairly good health, or may suffer from the symptoms common in rickets, when it is noticed to make a slight crowing sound occasionally. This may be repeated at intervals without giving rise to any alarm, but it gradually becomes more frequent, and may then be induced by the exciting causes above mentioned; however, it often occurs during sleep, and, on the whole, more during the night than in the day-time. After a while the interruption to respiration, at first only indicated by the crowing, becomes more marked. Breathing ceases, the chest is fixed, the face becomes pale and livid, the head is thrown back, and the facial muscles are slightly twitched. In a short time the spasm yields, and the air enters with a loud crowing noise through the still imperfectly opened glottis; and the child in a few minutes more may return to its playthings. In the severest cases the glottic spasm is accompanied by convulsive rigidity of the hands and feet, known as *carpopedal contractions;* the fingers are bent into the hand, the thumb within the fingers, and the hand is flexed on the wrist; the legs are extended, the feet bent on the legs, the soles turned inwards, and the great toe widely separated from the others. General convulsions may be added to these. Occasionally, death takes place during a fit, from complete stoppage of the respiration; and as the crowing is really the signal that the spasm is relaxing, it will be seen that in the fatal cases death may occur quite silently.

Many opinions have been advanced and discredited as to the causation of spasm of the glottis in children. Semon points out that bilateral cortical centres for the adduction of the vocal cords in phonation have been shown to exist (Krause, Semon, and Horsley), and it is probable that their defective nutrition and consequent irritability may have a large share in causing the symptoms of this disorder.

Diagnosis.—The symptoms are very characteristic, and not easily confounded with any other. The absence of fever, the shortness of the attack, and the completely healthy condition between the attacks, distinguish it from laryngitis. It may be simulated by the presence of a foreign body (*see* p. 470).

Prognosis.—This is, on the whole, favourable. The child may, indeed, die in a severe attack, and friends should be instructed what to do before a doctor can arrive. But if fatal results can be warded off, the disease in time subsides, or yields to treatment and the improved condition of the child's health.

Treatment.—This has to be considered in relation to the general health of the patient, and the occurrence of the attacks. The child must be put immediately under the best possible hygienic conditions: fresh air, well-ventilated rooms, and improvements in its food, where this is insufficient or unsuitable (*see* Rickets); and attention to the bowels should be secured. Medicinally, cod-liver

oil, or cod-liver oil with malt extract, is of great value, and the bromide of potassium or ammonium may be given three times daily, in doses of 2 to 5 grains, according to the age of the child. If the attacks are slight, sponging the child from head to foot two or three times daily with cold or tepid water, according to the season, often quickly stops them. In the more severe fits, the head should be raised, the surface of the body and face slapped with a towel dipped in cold water, and ammonia or acetic acid held to the nostrils. Or the body may be immersed in warm water, and cold water poured over the head and face. The administration, when the child can swallow, of tincture of castor, or of musk (gr. 1½ with sugar and gum in a teaspoonful of water), tickling the fauces, a few whiffs of chloroform, and enemata of 20 to 30 minims of tr. asafœtidæ, have also been recommended. Even after apparent death, artificial respiration will sometimes restore the patient.

SPASM OF THE GLOTTIS IN ADULTS

This occurs more frequently in connection with laryngitis, œdema of the larynx, paralytic conditions, or the presence of foreign bodies; it may also be a danger in epilepsy, tetanus, hydrophobia, or chorea. The entrance of saliva or small particles of food or drink into the larynx may cause most dangerous spasm; and a certain amount is often induced by the application of medicated solutions to the mucous membrane of the larynx. Apart from these causes, it is most often the result of hysteria.

Treatment.—Inhalations of chloroform, amyl nitrite, vapor coninæ, or burning stramonium should be employed, if they can be obtained in time; otherwise tracheotomy may be necessary. The bromides may be given for recurrent attacks.

CHRONIC INFANTILE STRIDOR

(Congenital Laryngeal Stridor)

Infants are occasionally the subjects of a laryngeal disorder, in which the breathing is accompanied by a peculiar croaking sound. This is generally first heard soon after birth, is continuous for long periods, perhaps all day and night, but may be absent for a few hours at a time. The croaking takes place with inspiration, and is either a rough rhonchal sound, or more clear and musical; expiration is either quite normal or rattling, as if from accumulated mucus; the cough and cry are, as a rule, normal. There may be a little sucking-in of intercostal spaces, but there is rarely any lividity. In some cases the noise is constant during sleep, in others it is absent. It is generally worse when the child is lively or excited. It subsides as the child grows older, but I have known it still present at two and a half years. The child appears to be in other respects quite healthy.

Both during life and after death the glottic aperture is seen to be extremely narrow, the epiglottis being folded on itself, and the ary-epiglottic folds almost in contact. This, however, is only an exaggeration of the normal condition of the infant's larynx, and Dr. Paterson has shown, by direct observation in five cases, that the stridor was due to the remarkable downward and forward movement of the soft posterior wall of the glottis, and its vibration in this position.

The obstruction caused by the deformity diminishes as the parts develop. No direct treatment is of any avail. Tracheotomy might be necessary in the rare event of life being threatened by asphyxia.

ANÆSTHESIA OF THE LARYNX

This occurs in diphtheria, in bulbar paralysis, in locomotor ataxy and general paralysis, and from injury to the vagus or superior laryngeal nerve. It is recognised by the insensibility of the laryngeal mucous membrane when touched with a probe, introduced with the help of the laryngoscope. It is often accompanied by dysphagia from particles of food entering the larynx, the result, according to Mackenzie, of paralysis of muscles supplied by the superior laryngeal nerve, *i.e.*, those which depress the epiglottis and close the upper aperture of the glottis during swallowing. Anæsthesia from diphtheria generally recovers; the prognosis is generally bad in progressive bulbar paralysis and allied conditions, as food is apt to get into the lungs, and set up pneumonia.

The Treatment should be by means of galvanic and faradic applications. Strychnia may be given internally, and dysphagia may necessitate feeding with the œsophageal tube.

DISEASES OF THE TRACHEA

TRACHEITIS

Inflammation of the trachea arises from circumstances similar to those producing laryngitis. Acute catarrhal tracheitis frequently accompanies laryngitis and bronchitis, but is masked by the symptoms which they produce. Occasionally it exists alone. It then produces cough, often hacking, perhaps violent or paroxysmal, with some amount of expectoration. With the laryngoscope, the mucous membrane may be seen to be congested, and ulcers are sometimes observed. With the stethoscope, mucous râles may be heard in the trachea; but the swelling of the mucous membrane and the mucous accumulation are not generally sufficient to cause much dyspnœa. The patient requires treatment similar to that

used in bronchitis—warm temperature and avoidance of exposure. Troublesome cough may be relieved by insufflations of morphia ($\frac{1}{16}$ to $\frac{1}{8}$ gr.) or bismuth nitrate ($\frac{1}{8}$ to $\frac{1}{4}$ gr.); and expectorants, such as squills and ipecacuanha, steam or benzoin inhalations, and the application of mustard to the upper part of the sternum, are of service.

The trachea is attacked by *diphtheria*, spreading from the larynx. Croup was at one time supposed to be mainly a tracheitis (*cynanche trachealis*), but it is now recognised that membranous inflammation of the trachea descends from the larynx.

Tubercle of the trachea occurs occasionally in association with tubercle of the larynx; ulceration follows the deposit of tubercle in the mucous membrane or submucous tissue. The ulcers are more common on the posterior wall, and usually measure from two to four millimetres, but may reach ten millimetres in diameter. The symptoms due to tracheal tubercle are generally masked by those to which simultaneous disease of the larynx or lung gives rise.

Syphilis, in its secondary and tertiary stages, also affects the trachea, producing in different cases congestion, condylomata (rarely), and superficial ulcers. The most important change, however, is stricture. The trachea is affected most often at its lower end, less commonly at its upper end; and the stricture may consist simply of a narrowing at one spot, or a considerable length of the trachea may be reduced in calibre. The mucous membrane is raised into bands and ridges, which have been regarded as cicatrices of former ulcers, possibly preceded by gummata; but German pathologists look upon the thickening as a direct result of the syphilis, and the ulceration as secondary. In later stages the cartilaginous rings have been exposed, necrosed, and expectorated or absorbed. The stricture can be sometimes seen below the glottis by means of the laryngoscope. For the symptoms, diagnosis, and treatment of stricture, *see* below under Tracheal Obstruction.

NEW GROWTHS IN THE TRACHEA

The trachea is remarkably little subject to primary new growths, whether benign or malignant. When present they give rise to dyspnœa, and may be recognised, possibly, by the use of the laryngoscope. More frequently cancer of the œsophagus or of the mediastinum grows into the adjacent trachea, narrowing its channel and producing symptoms of stricture. Where it spreads from the œsophagus, it is preceded by dysphagia; but tracheal symptoms may be the first indication of cancer of the mediastinum. Another way in which tumours affect the trachea is by simply compressing it from outside.

As the chief symptoms in all these cases depend on the reduction of the calibre of the trachea, and as this may be due to other

causes, besides such tumours, it will be well to consider separately the pathology and clinical features of tracheal obstruction.

TRACHEAL OBSTRUCTION

The causes may be grouped under three heads: (1) compression from without; (2) changes in the walls of the trachea itself (stricture); (3) foreign bodies within it.

Compression of the Trachea.—The most common causes are mediastinal new growths, aneurysm of the aorta or large vessels, enlargement of the thyroid body, and malignant tumours in the neck. Cancer of the œsophagus may also compress the trachea, but soon invades it, so that perforation takes place between the two tubes. Occasionally in children caseation and suppuration of the bronchial glands may lead to their enlargement, by which the trachea is compressed; and if the abscess bursts into the trachea, pus or portions of caseous glands may be expectorated. A mediastinal abscess, arising in any other way (as from caries of the spine), the dilated left auricle in cases of mitral constriction, and in children an enlarged thymus, are occasional causes of tracheal compression.

Stricture.—The chief cause is syphilis, which has been already considered.

Foreign bodies are rarely retained in the trachea, but commonly fall into one or other bronchus, though they may be driven up and down the trachea by the respiratory currents.

Symptoms.—The most important are dyspnœa and stridulous breathing; they are often accompanied by cough, and the expectoration of thin frothy mucus. The voice is unaffected, or it is feeble because the obstruction weakens the current of expired air. The chest is resonant, but vesicular murmur is faint, or drowned by the noise of the stridor. Other symptoms accompanying tracheal stenosis are due to the lesion which causes it, and these may be at first entirely absent in a case of aortic aneurysm or deeply-seated mediastinal tumour.

It is important to note that when the tracheal stricture or compression has reached a certain limit, the patient becomes liable to sudden attacks of aggravated dyspnœa with cyanosis. From a few of these paroxysms he may recover: but in the third or fourth or a later one he will probably die.

Diagnosis.—This has to be made (1) between obstruction in the trachea and obstruction in the larynx; (2) between the different causes of tracheal obstruction.

The laryngoscope will show at once the absence of laryngeal disease, and it may show the presence of tracheal stricture, or of tumour or aneurysm compressing this tube. This should be determined, if possible, before the occurrence of the paroxysms above mentioned, in which laryngoscopy may be difficult; and which, moreover, may wrongly suggest laryngeal spasm, and lead to a hasty and useless tracheotomy. There are some differences in the

effects of laryngeal and tracheal obstruction. One is the fact noticed by Gerhardt, that in laryngeal obstruction the larynx is moved extensively up and down in the neck during respiratory movements, whereas in tracheal obstruction it moves but slightly. In laryngeal obstruction the head is thrown back; in tracheal obstruction it is often bent forward. If the laryngeal obstruction is due to abductor paralysis, the stridor is chiefly inspiratory, whereas in tracheal obstruction there is generally some stridor with expiration. But in other cases of laryngeal obstruction the stridor occurs with both respiratory acts. Auscultation of the trachea is certainly deceptive, as the loudest stridor is heard over the larynx even when the stenosis is in the trachea. The point is of practical importance, because laryngeal obstruction may be relieved by tracheotomy, but tracheal obstruction rarely so; and it is desirable to spare the patient an operation of this nature when it can do no possible good. But a new growth or aneurysm in the neck or upper part of the chest may produce the two obstructions, namely, one directly, by pressure on the trachea, the other indirectly, by pressure on the recurrent laryngeal nerves so as to cause abductor paralysis.

The recognition of the cause of tracheal obstruction depends upon collateral symptoms. Any source of compression would probably involve other organs and thus cause dysphagia, obstruction of the veins of the head, neck, or arm, pressure on corresponding nerves, and dulness under the sternum, or at the upper part of the chest on one side. On the other hand, stricture due, as already stated, to syphilis must be free from such symptoms; but an aneurysm of the aorta may compress the trachea without, at first, any other symptom by which it can be recognised. For the diagnosis of mediastinal growth from aneurysm, the reader is referred to Diseases of the Mediastinum. The Röntgen rays may in either case render some assistance.

Prognosis.—This is very unfavourable, the commoner causes being little amenable to treatment; but the rare cases of abscess compressing the trachea may recover on the bursting of the abscess.

Treatment.—The indications are (1) to remove the cause, if possible; (2) to open the trachea below the obstruction where this is in the upper part; and (3) to relieve symptoms and secondary results. A diseased thyroid or hypertrophied thymus may be removed, and enlarged glands or growths in the neck; and abscesses, where accessible, may possibly be opened; but such opportunities are infrequent. If an aneurysm is diagnosed, the treatment for that condition should be employed; and for obvious stricture, active antisyphilitic treatment by means of mercury and potassium iodide. The latter drug should be employed in any case which does not present sufficient data for a positive diagnosis as to the cause of the obstruction. In the case of a foreign body, tracheotomy should be performed, and then efforts to dislodge it should be made by inversion of the patient, shaking, &c.

DISEASES OF THE BRONCHI

BRONCHITIS

Ætiology.—Bronchitis, or inflammation of the bronchi, occurs at all ages, and may arise from a variety of causes, amongst which the most frequent is exposure to cold or wet, or both combined. Under such circumstances it may be associated with simultaneous inflammation of the larynx and nasal mucous membrane, or the inflammation may commence in the latter, and spread downwards to the bronchi. Another cause is contact of the bronchial mucous membrane with irritating vapours, as air carrying solid particles, such as dust, fog, or the air of mines and of certain manufactories. Bronchitis may also be set up by the presence of foreign bodies actually in the bronchial tubes: this is comparatively rare, but blood effused into the tubes may act in this way, and it constantly occurs as a result of the deposit of tubercle or cancer in the substance of the lung. Certain infectious diseases, already described, are frequently accompanied by bronchitis—namely, typhoid fever, measles, scarlet fever, diphtheria, influenza, and whooping-cough; and it often occurs in Bright's disease.

Among causes that may be called predisposing are age, habits, the general health, and preceding conditions of the lungs and heart. Bronchitis is especially prevalent amongst infants, young children, and elderly people; whereas young adults and the middle-aged are much less subject to it. Habits of luxury, confinement to warm rooms, and undue wrapping up, render the subject liable to contract bronchitis on comparatively slight exposure; and those in weakly health, or depressed from insufficient food, exhausting occupations, or bad sanitary conditions, easily acquire it. Heart disease, impeding the circulation in the lungs, and previous attacks of bronchitis—all the more if they have left behind them emphysema or dilated tubes—dispose to the ready occurrence of the disease. Some other conditions of ætiological importance are those which involve constant exposure to the exciting causes, such as residence in towns, in cold, damp, and changeable climates, employment in mines, in wool and steel manufactures, and other such industries. Bronchitis is much more common in winter than in summer.

Pathology.—The mucous membrane is the part most affected, but in severe or prolonged cases the submucosa is involved, and, rarely, the cartilages of the bronchial tubes, and adjacent parts of the lungs. The first effect is increased vascularity and swelling of the mucous membrane, and after a short time a free secretion from the surface takes place. This catarrhal secretion is provided (according to Ziegler), partly by the blood-vessels, and partly by the epithelial cells and mucous glands in the large divisions. It consists

chiefly of mucus, and contains leucocytes and shed epithelial cells. In later stages the secretion becomes more and more opaque from the presence of increasing numbers of leucocytes, extravasated, according to most writers, from the blood-vessels, but according to others (Socoleff, Hamilton), produced by germination from flat cells in contact with the swollen basement membrane after the superficial layers of the epithelium have been shed. The secretion may also contain cells in a state of fatty degeneration, or cells containing particles of soot or dirt derived from the inspired air.

Sometimes the smallest tubes at the base of the lungs are filled with thick green pus. If the superficial part of the base of the lung be sliced off, and the exposed section be squeezed, minute drops of pus will be found to ooze freely from the cut surface. The condition is one of *capillary bronchitis*, or *bronchiolitis*. Fraenkel describes a bronchiolitis fibrosa obliterans occurring in workmen exposed to irritating air or dust: the bronchioles are obstructed by acute or subacute growth of connective tissue.

If, in ordinary catarrhal cases, the inflammatory process persists long enough, the fibrous coats of the bronchi become thickened, and infiltrated with leucocytes; the muscular fibres are atrophied by pressure; and the cartilages and mucous glands disappear from the same cause. Ultimately, in many cases, the bronchial tubes become dilated, and form fusiform or cylindrical wide channels, often reaching the surface of the lung (*bronchiectasis*).

As a result of bronchitis, the lung itself undergoes important structural changes. Acute bronchitis leads to *lobular collapse* and *broncho-pneumonia;* chronic bronchitis is followed by *vesicular emphysema*, and sometimes by *chronic interstitial pneumonia*. The last three will be spoken of separately.

Lobular collapse occurs in isolated lobules, when the bronchial tubes leading to them are blocked with mucus; since when a tube is plugged, the retained air, being stagnant in contact with the pulmonary capillaries, undergoes absorption, just as air is absorbed which has escaped into the subcutaneous cellular tissue. And, no doubt, eventually, the air will get absorbed from any portion of lung in which there is no movement, even without obstruction of the bronchial tube which leads to it.

Bacteria.—Streptococci, staphylococci, pneumococci, and influenza bacilli are the pathogenic organisms most frequently found in the secretions of bronchitis.

ACUTE BRONCHITIS

Symptoms.—Acute bronchitis begins with some malaise, and a sensation of tightness of the chest; and cough soon occurs. In mild cases the general disturbance may be but slight, and the illness is confined to cough, expectoration of mucus or muco-pus, with very little, if any, dyspnœa. But in severe cases there is slight fever—the temperature rising to 100° or 101°, the appetite

failing, the tongue furred, the bowels inactive and the urine scanty. The cough is at first hard and dry, and is often attended with pain behind the sternum and in the muscles of forced expiration from the strain put upon them. The expectoration is then but scanty, consisting of thin, frothy mucus, with, possibly, an occasional streak of blood. After a few days the cough becomes essier and looser, and the expectoration is more abundant, more opaque, and yellow or green, from the addition of increasing quantities of leucocytes. In slighter cases the expectoration is generally more in the morning, from the accumulation during sleep, and in towns this sputum is frequently black with pigment derived from the atmosphere. Dyspnœa is often considerable, with marked prolongation of expiration; the patient has to sit upright in bed (orthopnœa), and all the respiratory muscles are called into play. After a time the secretion of muco-pus becomes less, the cough is less frequent, and the symptoms gradually subside.

Physical Signs.—These are chiefly the result of the narrowing which the tubes undergo, and of the presence within them of the mucous or muco-purulent secretion. On *inspection* of the chest of one suffering from acute bronchitis, the breathing is seen to be quickened, the chest is symmetrical, and generally in a state of moderate over-distension. The accessory muscles of respiration are seen to be in strong action, and expiration is prolonged. *Percussion*, as a rule, yields a normally resonant note, but there is occasionally slight hyper-resonance from temporary over-distension of the air-vesicles; and rarely there is a little impairment of resonance at the base from accumulated secretion or from collapse. *Auscultation* shows that both inspiration and expiration are accompanied by *sibilant* or *sonorous rhonchi*, or various kinds of *râle*, or both combined (*see* p. 452). The coarser rhonchi are often felt by the hand placed upon the chest, and may be even heard by the patient himself, or those standing near him. Like the rhonchi, the râles may occur both in large and small tubes. The larger or coarser râles are heard with both expiration and inspiration, the finest râles only with inspiration. These sounds are not equally present in all cases or in all stages of the disease. In very mild cases they may be absent. In many cases rhonchi alone are present, and when both occur, the rhonchi appear first, the râles later: this is explained by the course of the changes in the bronchi already described. In severe cases the sounds are heard, variously mixed, over the whole chest, and may entirely drown the vesicular murmur.

When the smallest bronchial tubes are filled with purulent secretion in the form above described as a capillary bronchitis, the position is one of extreme danger. This occurs in people of middle and advanced age,* and is often the rapid termination of a chronic bronchitis, or occurs secondarily to other morbid processes in the body. It is characterised by severe dyspnœa, great lividity of the

* The "capillary bronchitis" of children is probably, in most cases, broncho-pneumonia.

face and extremities, and rapid exhaustion. The temperature is little, if at all, raised. The cough is at first frequent, with abundant expectoration of viscid glairy mucus, or muco-pus, or pus. The dyspnœa and lividity are the result of the imperfect aeration which the blood undergoes when the minute tubes are blocked with secretion; and this obstruction is shown by the retraction of the supra-clavicular, supra-sternal, and lower intercostal spaces with each inspiration. The chest is often resonant above, but the percussion note is impaired at the bases. Auscultation reveals small dull or crackling râles over the bases, back and front, almost, or entirely, masking the vesicular murmur. In later stages the patient becomes more livid and drowsy; he gradually assumes the recumbent position, generally on one side; the pulse is feebler and quicker; inspiratory efforts are less effectual; and the intercostal spaces are more sucked in. Expectoration gradually diminishes; and before death the disturbance of the cerebral circulation is shown in coma, often with a slight amount of delirium.

Clinically, Fraenkel's fibrous bronchiolitis closely resembles miliary tuberculosis.

Diagnosis.—The diagnosis of bronchitis itself presents few difficulties, as it is nearly always distinguished by sibilant or sonorous rhonchi. The dyspnœa and physical signs in *asthma* are like those of a very acute bronchitis; but the history of its occurrence and of former attacks will help to distinguish it. In the capillary bronchitis above mentioned, however, rhonchi are generally quite absent, and these cases are recognised by the lividity, drowsiness, absence of fever, and localisation of the râles at the two bases. Rarely, *obstruction* of one *bronchus* (*see* p. 497) may cause a stridor, which is mistaken for bronchitic rhonchus.

It remains to determine whether a bronchitis is primary, or secondary to such disorders as whooping-cough, measles, and typhoid fever, or is a part of acute miliary tuberculosis.

Prognosis.—The duration of bronchitis is from a few days to three weeks or more. In fatal cases it is from nine to twelve days, but children succumb more rapidly than young or middle-aged adults, in whom death rarely takes place from simple acute bronchitis. Capillary bronchitis is much more dangerous. Bronchitis complicating other diseases, such as cardiac lesions, the exanthemata, Bright's disease, or typhoid fever, presents essentially the same features, and, as it varies in all cases from very mild to the most severe forms, its prognosis must be considered in the same way as in the simpler forms.

Treatment.—In cases of moderate severity the patient should be placed in bed in a warm room; and much relief is often given if the air of the room be kept constantly moist by the steam issuing from a "bronchitis kettle." In the first stages of dryness and stuffiness, saline remedies (potassium citrate, liquor ammonii acetatis), with small doses of ipecacuanha ($\frac{1}{2}$ to 1 grain, or 5 to 7 minims of the wine), should be given. If there is much tightness of the

chest, counter-irritation by means of mustard-leaf, or a linseed-meal poultice sprinkled with mustard, should be employed. The diet should be light and nutritious. In children, counter-irritants should be used with care, but a thin poultice surrounding the whole chest (jacket poultice) is of great service. In children, also, an emetic (one drachm of vinum ipecacuanhæ, repeated in fifteen minutes if necessary) is sometimes of use to get rid of accumulated bronchial secretion; and the same much more rarely in adults. As cough becomes looser, and the sputum more abundant, expectorants may be more freely used—such as ammonium carbonate (5 grains every four hours), vinum ipecacuanhæ (5 to 10 minims), or tinct. scillæ (15 minims), or a combination of two or more of them. When the cough is very irritating, sedatives may be employed—morphia in small doses ($\frac{1}{8}$ grain or $\frac{1}{6}$ grain), compound tincture of camphor ($\frac{1}{2}$ drachm), heroin ($\frac{1}{20}$ grain to $\frac{1}{10}$ grain), tinct. belladonnæ (10 minims), or potassium or ammonium bromide (5 grains). But they must be used with great care if there is much lividity, as they may dangerously depress the respiratory and cardiac centres under these circumstances. Cases accompanied with much spasm of the bronchial tubes may be benefited by tinct. lobeliæ (15 minims), ether (15 minims), tinct. cannabis ind. (10 minims), or potassium iodide (5 to 7 grains). Chloral in small doses (5 to 7 grains) has also been recommended. In capillary bronchitis, stimulants must be given early; and the drugs of most value are ammonium carbonate, senega (as tincture or infusion), oil of turpentine (20 minims), and lobelia. Sedatives must be given with the greatest caution or entirely avoided, for the reasons given. In all severe cases, oxygen inhalations may render valuable service.

CHRONIC BRONCHITIS

This occurs as a sequel of the acute form, or begins in exposure or chill in those who have had a previous attack. It is common in elderly people, often appearing in successive winters and subsiding with the approach of summer. After a time it may become continuous throughout the year.

Symptoms.—The main features of chronic bronchitis are not essentially different from those of the acute form: cough, dyspnœa, expectoration of mucus, muco-pus, or pus, with the rhonchi and râles already described. But there is an absence of fever and the constitutional disturbance which occur in acute attacks; and after long continuance, secondary results of a permanent kind are produced. In the lung itself, emphysema and dilatation of the tubes (*bronchiectasis*) take place, and these will be described later. But the effects are felt beyond the lung: the deficient aeration of the blood, which results from long-continued bronchitis, impedes the pulmonary circulation, and the right heart becomes hypertrophied; or it dilates, and thereupon the general venous system suffers, so that œdema of the lower extremities, congestion of the liver, ascites, and albumi-

nuria occur. Under such circumstances, tricuspid regurgitation often takes place, with its characteristic murmur (*see* Diseases of the Heart). Long-continued and severe chronic bronchitis has a serious effect upon the strength of the patient. In consequence of disturbed sleep, abundant expectoration, and impaired digestion, nutrition fails, and there may be considerable emaciation. In some cases, also, in this late stage, febrile reaction of a hectic type may set in. The varieties of chronic bronchitis commonly described are the following:

1. By far the larger number of cases come under the head of ordinary *winter cough*, occurring as above mentioned. The cough is variable, sometimes coming on in paroxysms, generally worse at night; and in the morning, also, there is often severe coughing to expel the secretions which have accumulated during the period of rest. According to the severity or extent of the affection, the expectoration may be slight in amount, thin, mucous, and frothy, and containing black pigment in the morning; or it may be yellow or yellowish-green and muco-purulent, with very little air; or it may be entirely airless, liquid, green pus. The sputa in this case generally run together in the vessel, and do not exhibit the *nummular* character common in phthisis. Microscopically, besides the abundant pus-cells, there are epithelial cells containing fat, and non-pathogenic micro-organisms. Blood is occasionally present in the expectoration, usually in the form of streaks, but rarely in masses, or in any considerable quantity.

2. *Dry bronchitis*, or *dry catarrh*, is a form of chronic bronchitis in which there is very little secretion. The cough is frequent, violent, and prolonged, so that extreme congestion of the face occurs; but there is either no sputum at all, or only a small quantity of tough mucus. There is much soreness of the chest and considerable dyspnœa.

3. In *bronchorrhœa* the expectoration is exceptionally abundant, and either thin, clear, and transparent, or thick and ropy; it usually contains but little air. As much as four or five pints may be expectorated in twenty-four hours, large quantities being brought up at a time with comparatively trifling effort.

4. *Putrid* or *fœtid bronchitis* is characterised by very offensive sputum, which is not connected with gangrene of the lung, but mostly occurs in cases where the tubes are dilated. It will be described more fully below.

Physical Signs.—The auscultatory signs are similar to those of acute bronchitis, but as chronic bronchitis of long duration is commonly accompanied by emphysema, the signs of this disease may be also present (*see* p. 502). In cases with much accumulation of secretion the bases are dull; in such cases, also, there are abundant râles at the bases; but generally the physical signs consist of sonorous or sibilant rhonchi, more prolonged during expiration than inspiration; and of these the coarser and louder may be felt by the hand placed on the surface.

Diagnosis.—This is generally simple; the history, with the symptoms and physical signs, being sufficiently characteristic. There is occasionally a difficulty in excluding chronic phthisis, for phthisis is often accompanied by bronchitis. Here the occurrence of febrile reaction, of hæmoptysis, of rapid wasting, and the greater intensity of the physical signs on one side or at one apex, would be in favour of phthisis, and confirmation may be obtained by examination of the sputum for bacilli, by the use of X-rays, or of tuberculin (*see* Phthisis). It is important to recognise the cause of chronic bronchitis in cases, such as Bright's disease, where it is secondary.

Prognosis.—Though chronic bronchitis frequently shortens life, many people live to an advanced age in spite of it. It is mostly affected by the season in a marked manner, and patients are often practically well during the summer, and again get ill in the winter; but they are worse with each succeeding winter, and finally may be carried off during an exceptionally severe season, or during the cold fogs of towns, or during east and north-east winds elsewhere. On the other hand, if they can be protected from this unfavourable weather by confinement to the house, or better by residence in a warmer climate, they may keep their bronchitis within limits, and postpone the fatal termination for years. Its ill-effects will, however, vary with the amount of secretion and with the rapidity with which the secondary results—emphysema, dilated tubes, and dilatation of the right heart—are developed.

Treatment.—In chronic bronchitis, as already indicated, the patient must be carefully guarded from exposure, and kept as far as possible in a uniform temperature of 60° or 65°: a moderately nutritious diet should be allowed. The medical treatment consists mainly in the use of different kinds of expectorants. Ammonium carbonate, squills, ipecacuanha, and senega may be given in ordinary cases, but particular indications have to be followed in some instances. Ipecacuanha, potassium iodide, and apomorphine hydrochloride ($\frac{1}{10}$ grain) are of use in those with hard dry cough; potassium or ammonium bromide where the cough is very irritating. In such cases, opium or morphia may have to be used, and it often gives much relief; but it must be used in small doses at all times, and withheld in cases where cyanosis is at all advanced. Where the expectoration is abundant, the balsams of Peru (20 minims suspended with $1\frac{1}{2}$ drachms of honey) and tolu should be given, or ammonium chloride (5 to 20 grains); and the tendency to emaciation from the continuous drain should be met by the use of tonics at the same time. In cases where spasm of the tubes is liable to occur, lobelia, Indian hemp, sulphuric ether, spiritus chloroformi and stramonium are of value, and potassium iodide has been recommended for the same purpose. Turpentine, terebene, and copaiba are often beneficial in cases with free secretion. Good may be derived from inhalations of the vapour of ammonium chloride, or of steam, or the use of sprays charged with succus conii, tr.

benzoin co., and iodine or tar in the case of profuse expectoration. Benefit is also obtained from applications to the chest: linseed-meal poultices may be employed more or less continuously; and mustard plasters, turpentine stupes, and tincture or liniment of iodine, at longer or shorter intervals. Dry-cupping may also be of value. If the bronchitis can be referred to any constitutional disease, this should of course, at the same time, be treated—for instance, gout by the exhibition of alkalies and colchicum. Many cases require tonics, such as quinine and cod-liver oil. It is desirable to see that the bowels are freely opened; and in cases of long standing, where the right side of the heart is dilated, the various secretions should be kept free, and the heart's action assisted by digitalis or strychnine, as under corresponding conditions in valvular disease.

The most satisfactory results are got in some cases from residence in the south of England or abroad. Torquay, Bournemouth, Penzance, Mentone, San Remo, Cannes, Arcachon, Canary Islands, Madeira, and the Nile (Assouan) are the places most frequented.

FŒTID BRONCHITIS

The characteristic feature of fœtid bronchitis is the offensive odour of the sputum. It occurs most commonly as a result of bronchiectasis, when the secretions are allowed to accumulate in the dilated bronchial tubes, and so undergo decomposition. But it may arise under other conditions—for instance, in acute bronchitis —and the cause is probably in most cases the entrance of bacteria, by means of inspired air. Several observers have found bacilli in the sputum of fœtid bronchitis.

Symptoms.—The sputum is abundant and rather thin, and in the sputum-vessel it often separates into three layers, of which the uppermost is muco-purulent and frothy, the middle a thin seromucous fluid, and the lowest a thick layer of pus containing the bodies known as Dittrich's or Traube's plugs. These are whitish-gray or dirty grayish-yellow, varying in size from a millet-seed to a bean. Under the microscope they are seen to consist of pus-corpuscles, detritus, bacteria, bundles of fine acicular crystals of palmitic and stearic acids, and twisted threads of leptothrix; but neither lung tissue nor specific organisms occur in the sputum. The chemical contents of the sputum are acetic, butyric, and valerianic acids, leucin, tyrosin, sulphuretted hydrogen, and methylamine. The odour is very offensive, putrid, and at the same time somewhat sweet; not only the sputum, but the breath of the patient is charged with it, so that none can stay near him, and it pervades the atmosphere of the room. The onset of the putrid change may be accompanied by febrile reaction, prostration, and loss of appetite; and the septic condition may extend to the pulmonary tissue, so as to cause lobular pneumonia or gangrene. But fœtid bronchitis may become chronic, with only occasional attacks of pyrexia; with loss of appetite, nausea, and indigestion, but

otherwise no serious impairment of health. Painful swellings of the joints have also been recorded. Some patients recover completely.

Pathology.—In fatal cases one generally finds dilatation of the bronchial tubes of old date, with intense injection and ulceration of the mucous membrane.

Diagnosis.—The distinction from gangrene is difficult, and all the more so as the two may coexist. The physical signs of fœtid bronchitis are mostly those of bronchitis with dilated tubes—namely, fine or medium râles; while in gangrene one gets more often signs of consolidation or excavation. The presence of lung-tissue in the sputum would be in favour of gangrene, but it is not constantly found in that condition.

Treatment.—This must be of a supporting and stimulating kind, while locally we endeavour to lessen the decomposition and diminish the fœtor by antiseptic inhalations. Carbolic acid, creosote, turpentine, thymol, eucalyptol, and tincture of iodine may be used, either inhaled from the surface of hot water, or administered more continuously by means of a respirator (*see* Treatment of Phthisis). Counter-irritants and blisters may also be of value.

PLASTIC BRONCHITIS

(*Fibrinous Bronchitis. Croupous Bronchitis*)

This affection is characterised by the expectoration of casts of the bronchial tubes. The sputum is generally in the form of a rounded mass, covered with mucus or blood, and, when frayed out in water, one sees a more or less perfect branching cast of a portion of the bronchial tube system. The cast is not generally thicker than a goose-quill, and varies from one and a half to two and a half inches in length, and only rarely reaches four or five, or even seven inches. It has a gray or whitish-yellow colour, and consists of concentric laminæ, which do not usually fill up the lumen of the tube, so that the casts are not solid, except those from the smallest tubes. Under the microscope the cast has a fibrillated structure, with numerous imbedded leucocytes, streptococci and staphylococci, hæmatoidin crystals, Curschmann's spirals, and Charcot-Leyden crystals (*see* p. 495).

Ætiology.—The disease is extremely rare; it occurs in males more frequently than in females, and mostly begins between the ages of ten and forty; it has also been noticed to occur in different members of the same family.

The **Symptoms** preceding the expectoration of the casts may be very slight; indeed, the patient may appear to be in ordinary health; or there may be indications of bronchial catarrh, or a pyrexial condition, with rigors suggestive of pneumonia. Then the patient is seized with violent attacks of coughing, often suffocative in character, with more or less pain or oppression at the chest, and

attended at first with no sputum, unless perhaps a little mucus. After a time—it may be a few hours, or as long as two or three days—a bronchial cast is brought up. Relief is generally at once afforded: the cough subsides or disappears. But it commonly recurs in a few hours, and casts may continue to be expectorated, at intervals of a day or so, for several days, when the patient gradually gets quite well. Hæmoptysis occurs in some cases, usually after the expulsion of the cast. The physical signs are attributable to the obstruction of the tube or tubes. The vesicular murmur is commonly deficient; and there may be either slight increase of resonance over the area of lung corresponding to the obstructed tube, or, on the other hand, dulness from collapse of the same portion. The movements of one side of the chest may be impeded if tubes are extensively blocked; and râles, clicks, or flapping sounds are sometimes heard as the casts are becoming loosened.

Prognosis.—The disease is rarely fatal, except from complications; but it recurs at irregular intervals over a period of several years.

The **Treatment** hitherto tried has been unsatisfactory. Iodide of potassium in full doses is credited with some success; mercurials, tartar emetic, alkalies and their carbonates, have been at different times used. Inhalations of steam and tar vapour, and sprays of lime-water and of alkaline carbonates, are also recommended; and an emetic is said sometimes to be of value.

BRONCHIECTASIS

Bronchiectasis or dilatation of the bronchi may occur in connection with many diseases of the lungs. Some of these act in virtue of being inflammations, such as bronchitis, chronic pneumonia and phthisis, during the course of which bronchial tubes may dilate. Acute broncho-pneumonia, especially in children, is often a cause of bronchiectasis; and lobar pneumonia and pleurisy are sometimes followed by dilatations, the position of which is determined by the primary lesion.

In other cases the cause is primarily mechanical: for any gradually increasing and persistent obstruction of a large bronchial tube is likely to be followed by dilatation of the smaller bronchi proceeding from it. Thus aneurysms pressing on a bronchus, cancer pressing on or growing into it, and syphilitic stenosis are frequent causes of bronchiectasis. A foreign body impacted in a bronchus almost inevitably leads to bronchiectasis.

Rarely the condition is congenital, and it is not unfrequently seen in quite young persons, when the cause may be unknown or forgotten.

The effect of a continued bronchitis is to loosen the tissues of the bronchial wall, and lead to more or less atrophy of the muscular fibre and of the cartilages. The bronchial wall is then likely to

yield to the pressure of the air in coughing efforts and other strains, or to the pressure of the secretion as long as it is retained. Retained secretion, and septic influences on the bronchial structures, explain the dilatation following upon obstruction. In ordinary cases the change affects the medium and smaller tubes; they are dilated into somewhat irregular cylindrical tubes (*cylindrical bronchiectasis*), and can be traced with the greatest ease nearly to the surface of the lung; they are more common in the lower lobes than in the upper. Though the process is usually a chronic one, an acute bronchiectasis of numerous smaller tubes (*bronchiolectasis*) throughout the lung is occasionally seen after an acute catarrhal bronchitis.

In other cases the dilated tubes are ovoid or globular (*saccular bronchiectasis*). In this variety a number of smooth-walled cavities, from the size of peas to marbles, or larger still, are found in the lungs; their walls are thin, and present generally no trace of the muscular tissue or cartilage of the healthy bronchi; a small bronchus may often be found opening into the cavity. Sometimes there are bands running along the walls; sometimes the surface is ulcerated.

This kind of cavity is frequently associated with the extensive fibroid changes found in chronic pleurisy and fibroid phthisis, and the contraction of the fibrous tissue tends to the enlargement of the cavities. They are situated much more commonly at the base and the middle of the lungs than at the apex.

Bronchiectasis is often limited to one lung, especially when due to bronchial obstruction, to a foreign body, or to acute pneumonia or pleurisy. If both lungs are affected either the lesions are not extensive, or one lung is very much more involved than the other. Emphysema may accompany bronchiectasis.

Symptoms.—In cases of moderate cylindrical dilatation associated with bronchitis or emphysema, the symptoms will be lost in those of the primary disease. But in larger dilatations and in the saccular variety the bronchiectasis is the prominent fact in the case, and the secretion from the cavities and the fibrosis and cavitation of large portions of the lung are productive of definite symptoms and physical signs.

The patient need not be emaciated, is generally free from fever, and may be inconvenienced by little besides dyspnœa, cough, and expectoration; but he is often cyanotic, and sometimes extremely so; the fingers are clubbed (*see* p. 531), and in course of time the effect of the pulmonary lesions upon the right heart will cause œdema of the feet, enlargement of the liver, and albuminuria.

The sputum is either purulent and airless, or muco-purulent, or fœtid, muco-purulent and frothy, like that of fœtid bronchitis. When there are one or two large saccular cavities the sputum is often expectorated in a characteristic manner. The secretion collects for some time—it may be two or three hours—in the dilated tubes, without exciting cough; then either from its quantity, or because the patient moves about, turns over or sits up in bed, the secretion flows over into an adjacent healthier tube, coughing is

excited, and some ounces of muco-purulent secretion are all at once expectorated. In some cases, hæmoptysis is both frequent and moderately abundant.

Physical Signs.—These differ according to the character and size of the dilatations, their distribution in the lung, and the amount of consolidation or fibrosis of the intervening lung. In some cases, a large portion of one base, or even the whole of one side of the chest, presents coarse, creaking, and crackling râles, obscuring the respiratory murmur, but without dulness or pronounced limitation of movement. In other cases the condition is similar, but in addition there is, at one or more spots, an area where bronchial or cavernous breathing, with bronchophony and good transmission of whispered voice, is heard. These signs indicate a cavity near the surface, or one surrounded by condensed lung tissue.

In other cases the physical signs are present in only one region of the chest, generally either the base, or the middle zone, and rarely the apex. There are dulness, bronchial or cavernous breathing, bronchophony, pectoriloquy, and some râles. The breath-sounds and the râles vary with the amount of secretion in the cavity, in the same way as do those of phthisical cavities, and the sound ascribed to *post-tussive suction* may be heard in conditions similar to those of phthisis (*see* p. 453). In extreme cases, or when fibrosis is advanced, the condition resembles chronic pneumonia. Retraction of the chest takes place, the heart is drawn in a horizontal direction towards the diseased lung; and the opposite lung becomes compensatorily emphysematous. In late stages the right side of the heart dilates, and œdema and venous stagnation ensue.

Diagnosis.—The disease is readily confounded with *chronic phthisis*. The chief points of distinction are that in bronchiectasis the physical signs are not confined to, nor most marked at the apex, the patient is not febrile, nor ill and emaciated in proportion to the extent of the local mischief—indeed, he is often for a long time well nourished—and there are no tubercle-bacilli in the sputum. The pronounced cyanosis and clubbed fingers might suggest *congenital disease of the heart;* but the physical signs show that the lung and not the heart is primarily at fault. A basal bronchiectasis may be hard to distinguish from an *empyema* discharging into the bronchi. The history may help; and hæmoptysis is in favour of bronchiectasis. Exploration might yield pus in either case.

Prognosis.—As compared with phthisis it is good; patients often live for years with but slow advance in the local conditions; but they are liable to dangerous complications, such as pneumonia, gangrene, septicæmia, and pyæmia, with formation of abscess in the brain or liver.

Treatment.—The patient requires support by means of tonics, such as iron, quinine, and cod-liver oil, and should be placed under the best hygienic and climatic conditions, on the same principles as in the treatment of phthisis. Locally the object should be to assist the evacuation and the disinfection of the secretions. Inhalations

of antiseptics (*see* Bronchitis) are of value; but other more thorough means have been tried, as, for instance, Poore's administration of garlic internally, by means of capsules containing 30 grains of chopped garlic; the daily inhalation for 15 to 60 minutes of the vapour of creosote in a closed chamber (Chaplin); and intra-tracheal injection of antiseptics. One commonly employed has been a solution of 10 parts menthol, 2 parts guaiacol in 88 parts olive oil, of which one drachm is used twice daily. Mendel recommends a solution in olive oil of 5 to 10 per cent. of eucalyptol and 5 to 50 per cent. of gomenol. He injects 3 c.c. at a time by means of a syringe, of which the nozzle is directed to the lateral wall of the pharynx, so that the fluid runs round into the back of the glottis, which by traction on the tongue is pressed against the pharyngeal wall.

ASTHMA

In asthma the patient suffers from sudden attacks of dyspnœa, which subside after a time, and recur at irregular intervals. The dyspnœa results from obstruction of the smaller bronchial tubes, probably due to spasm of the muscular fibres in their walls.

Ætiology.—One must consider, first, the causes of the disease itself—that is, of the tendency to suffer from the paroxysms of dyspnœa; and secondly, the various circumstances which may bring on an attack in those disposed to them. The disease is often transmitted hereditarily, but it may not in such cases show itself till an advanced age. In children, who contribute a large proportion of asthmatic cases, it may follow measles, whooping-cough, or bronchitis. Adenoid growths in the nose and naso-pharynx sometimes cause asthma. A neurotic origin is often noticed. Salter observed that it might alternate with epileptic attacks, while it is sometimes associated with neuralgia, angina, or other neuroses in the same person. Gout rather predisposes to asthma, and malaria and syphilis have been held responsible for some cases; moreover, it sometimes occurs on the subsidence of skin eruptions, such as eczema and lichen. The disease begins at all ages; it is twice as frequent in males as in females.

Amongst the exciting causes of an attack are a number of impressions upon the peripheral nerves, or more central disturbances of the nervous system; and the greatest variety exists as to the way in which, in different persons, the attack is brought on. Particular climates or atmospheres, cold air, the close atmosphere of a badly ventilated room; particles of dust or fluff; smoke; the odour of hay, of certain flowers, or of ipecacuanha; or the emanations from some animals—namely, cats, rabbits, dogs, horses, &c.—may at once produce it. Diet has an important influence; any overloading of the stomach may set up an attack in some persons, but a late meal is especially likely to do it; or certain articles of diet have to be carefully avoided. Constipation and uterine

troubles occasionally act as irritants. Emotion, anger, and fright are instances of cerebral disturbances causing asthma.

Symptoms.—Sometimes there are *premonitory* indications, such as a general sense of discomfort, drowsiness, gaping, itching under the chin, sneezing and coryza, or the passage of much pale limpid urine. But the attack is often quite sudden, commencing in the early morning between two and four o'clock, though the patient may have gone to bed apparently quite well. He wakes up with a sense of dyspnœa, so that he has to sit up in bed, or gets out and opens the window to let in more air. The breathing is soon so difficult that he has to call in the aid of all the accessory muscles of respiration; he grasps with his hands the sides of his bed, the arms of a chair, the mantelpiece, or the edge of a table, to give a firm support for the muscles which pass from the upper extremities to the chest. The chest, however, is nearly fixed in a condition of inspiration, and there is very slight movement. The respiration is often quite slow, but occasionally rapid; the most noticeable feature is the extraordinary length of expiration, which is accompanied with a loud wheezing, audible at a distance. The chest is somewhat over-resonant; the inspiratory murmur is scarcely audible, or accompanied with a little sibilant rhonchus, while with expiration is heard the loud rhonchus just mentioned. With this the patient's distress is very great; the face gets cyanosed, the eyes are prominent, the conjunctivæ suffused, and the whole attention of the patient is absorbed in the attempt to get a proper interchange of air in the chest. Usually there is no pyrexia. After a time—it may be two or three hours—he begins to cough, and expectorates some thin, transparent mucus, which may be mixed with a little blood; then the breathing becomes easier, the cyanosis is less, gradually the whole trouble subsides, and the patient falls asleep.

The sputum often contains, besides cylindrical or ciliated epithelium, two peculiar constituents—namely, Curschmann's spirals, and octahedral crystals. The former are yellowish-green or gray particles, made up of threads of mucus. Under the microscope they are seen to be spirally twisted fine or coarse fibres, and there is often in the middle one transparent fibre; they are probably formed in the finer bronchial tubes. The octahedra may be present in the spirals; they are known as Charcot-Leyden crystals, and consist of phosphate of spermin. Similar crystals are found in the blood in leuchæmia, and sometimes in the fæces in dysentery, typhoid fever, and other conditions. The eosinophile leucocytes of the blood are increased in number.

Each attack of asthma may last from two or three hours to as many days; their recurrence, at longer or shorter intervals, is a good deal determined by the exciting causes—that is, a careful patient, who knows how to avoid what will bring on his attacks, may escape for long periods. The duration of the illness is also very variable. Many of those who have it in childhood recover in adult age; but those who acquire it in middle age never

recover. The attacks themselves are rarely fatal, and the occasional occurrence of not very severe attacks is not prejudicial to health: but frequent paroxysms induce emphysema of the lungs, and ultimately attendant bronchitis, so that there is constantly more or less lividity, with the round shoulders, barrel-shaped chest, and laboured respiration which are observed in the midst of the paroxysms themselves. Life is thereby shortened, and the tendency to suffer from the severer forms of bronchitis is increased.

Pathology.—The attacks are clearly attributable to some obstruction of the minute bronchial tubes, and the prevailing view is that this obstruction is due to spasmodic contraction of the bronchial muscular fibre; hence the name "spasmodic asthma," to distinguish it from other kinds of dyspnœa, especially chronic bronchitis and emphysema, known popularly as asthma. But the disease has also been attributed to vascular or erythematous swelling of the bronchial mucous membrane, in which case it might be a very acute catarrh, or a vaso-motor neurosis allied to angeio-neurotic œdema. The sudden development and the rapid subsidence in many cases are in favour of its spasmodic origin.

Diagnosis.—This is not generally difficult, if the history and the character of the breathing and its onset be carefully studied. Sudden attacks of dyspnœa in cardiac disease, thoracic aneurysm, and laryngeal obstruction are those which are likely to resemble it most closely. Hysterical attacks may also simulate it.

Treatment.—Climate is one of the first things to be considered. A large number of patients can live in London and large cities free from paroxysms, who have them at once if they attempt to live in the country. Conversely, some can only live in the country, and have asthmatic attacks in town. In the same way sea-air may excite attacks in some and cure others. The facts with regard to any patient can only be ascertained by experiment.

Moderation and care in diet are the next points to consider. Food should be light and easily digestible; a heavy supper should not be taken; and particular foods should be excluded from time to time, such as cheese, pastry, pork, beer, to see if there is any one offender in this respect. If the trouble cannot be met in these ways, and the naso-pharynx presents no lesions for surgical treatment, potassium iodide should be given in doses of from 5 to 10 grains three times a day, continuously and irrespective of the attacks, for some weeks or months; and arsenic is similarly useful. A number of remedies have been used in the attacks, and many of them are decidedly effectual in lessening the severity of the dyspnœa and shortening the paroxysm. The most useful seem to be those which are inhaled, and so possibly act directly upon the bronchial tubes. The vapours of chloroform, ether, nitrite of amyl, iodide of ethyl, and turpentine may thus be used; but more lasting results are often obtained by the fumes from burning a paper saturated with nitre-solution and dried, or by smoking cigarettes made of chopped stramonium-leaves, or by the use of other preparations

containing stramonium. Some similar drugs may be given internally, such as nitro-glycerin, and nitrite of sodium—which also paralyse organic muscular fibre—and chloral, morphia, potassium bromide, antipyrin, extract or tincture of stramonium, belladonna, or lobelia. Adrenalin chloride has a good effect on asthma: 3 to 5 minims of a solution (1 in 1000) should be injected hypodermically at the beginning of an attack. Two or more remedies may be combined. Local applications may give some relief, such as mustard plasters or turpentine stupes; and, according to some, the application of iodine tincture on the side of the neck, over the course of the pneumogastric nerves. In chronic cases which resist treatment, general tonics, like quinine or cod-liver oil, may be of value, and the patient should be careful not to expose himself to cold unduly, in view of the secondary changes in the lung which supervene.

OBSTRUCTION OF THE LARGE BRONCHI

In their relation to the various causes of compression, the two main bronchi closely resemble the trachea, and much that has been said under the head of tracheal obstruction might be repeated here. Aneurysms and bronchial glands enlarged by malignant growth, or by caseation and suppuration, are the chief causes of compression: less commonly epithelioma of the œsophagus, gummata, abscesses, and even a dilated left auricle may press upon the bronchus. Stricture occurs from primary cancer of the mucous membrane, usually a columnar-celled carcinoma, and from syphilitic ulceration and scarring. Obstruction also results from an impacted foreign body. One or both bronchi, and perhaps at the same time the lower end of the trachea, may be compressed by growth or aneurysm. The special liability of the left bronchus to compression by an aneurysm of the arch of the aorta, under which it passes, is of importance. Foreign bodies more frequently fall into the right bronchus, because the dividing ridge between the two bronchi is somewhat to the left of the middle line, and hence objects falling down the centre of the trachea are directed into its right branch. They may be driven into the trachea during coughing, and fall back into the same or the opposite bronchus. If the object is impacted it causes a permanent obstruction, and may lead to hæmoptysis or ulceration and sloughing of the mucous membrane.

The permanent obstruction of a bronchus is followed by important changes in the corresponding portion of the lung, and the distal divisions of the bronchus. Ultimately in every case the *lung becomes collapsed*, because, when the interchange of air completely ceases, what remains is absorbed by the pulmonary vessels. In a rapidly complete obstruction, as from the entrance of a foreign body, this collapse occurs very early; but when the compression

takes place slowly, as in the case of an aneurysmal sac, there is at first *distension* of the lung with air, such that the heart may be pushed out of place, and the diaphragm forced downwards (G. N. Pitt). This is explained by supposing that the inspiratory muscles can suck in air, but that, on account of the obstruction, the elastic expiratory force of the lung is insufficient to expel the tidal air; and that when the expiratory muscles are called into play the bronchial tubes are compressed as well as the lungs, so that the discharge of air is still impeded.

On the bronchi beyond the seat of obstruction the certain effect of the narrowing is the occurrence of dilatation or *bronchiectasis*, which may develop in the course of two or three months.

The retained secretions, accumulating in the tubes, press upon their walls, and these, weakened by tissue-changes, induced by the septic condition of the secretions, necessarily yield. Accompanying the bronchiectasis, there is in course of time *fibrosis* of the lung, and thickening of the adjacent pleura.

Pus forms in the bronchiectatic cavities, and though expectorated from time to time, is likely to undergo septic changes or putrefaction, and contributes to the occurrence of septic pneumonia, gangrene of the lung, acute pleurisy or empyema.

Coupland has recorded a case of obstruction in which the dilated tubes communicated through one of the intercostal spaces with the subcutaneous tissue on the front of the chest, where an abscess formed containing offensive pus; and I have seen the left lung converted into one large sac containing pus, from the pressure of an aneurysm on the bronchus. Foreign bodies do not always confine themselves to producing mechanical obstruction, but they have occasionally set up diffuse suppurative pneumonia, or have worked their way to the surface of the lung, perforated the pleura, and caused pleurisy or pneumothorax. A fragment of tooth, accidentally detached during extraction, may cause hæmoptysis, and local signs over a small area, deceptively like phthisis.

Symptoms and Physical Signs.—These vary with the degree of obstruction: and since the opposite tube is often free, and thus only half the respiratory area is interfered with, the bronchus is often much more completely obstructed, before death occurs, than ever the trachea can be.

Dyspnœa is the only constant symptom at first, and only occasionally is there stridor; but stricture of either main bronchus may lead to the same paroxysms of asphyxia as occur in tracheal obstruction. When bronchiectasis has developed, cough, expectoration of offensive sputum, and febrile reaction become prominent symptoms.

The chief physical sign is the absence or extreme weakness of the vesicular murmur, which is in strong contrast with the supplementary breathing on the opposite side. This, in some cases for a time, may be the only physical sign, for the resonance may be perfectly normal. But in those cases in which distension of the

lung takes place, there will be hyper-resonance on percussion, with extension of resonance over the cardiac area, and evidence of displacement of the heart, so that the resemblance to pneumothorax may be very close. In these cases eventually, and in other cases much sooner, as the air becomes absorbed, there is dulness at the affected base, with diminished tactile vibration. This may go on to complete absence of breath-sounds, voice-sounds, tactile vibration, and percussion resonance. If considerable bronchiectatic cavities should be formed, the above physical signs may be, over one or other small area, replaced by tympanitic percussion note, cavernous breathing, moist or gurgling râles, and pectoriloquous voice-sounds.

The cases run a hopeless course, the dyspnœa increases, the temperature oscillates as usual in septic conditions, the patient becomes sallow and emaciated, and dies from exhaustion, or from the rupture of the aneurysm, or from septic pneumonia.

Diagnosis.—The combination of good resonance with nearly complete absence of respiratory sounds on one side of the chest is very characteristic of obstruction of the corresponding bronchus. When the obstruction is accompanied by stridor, it may be mistaken for bronchitis. Stridor from the above cause is persistent and uniform in character, arising from a single point of obstruction, whereas the rhonchi of bronchitis vary constantly in loudness, pitch, and position. Bronchitis further is more often bilateral.

In compression of the bronchus with a distended lung, pneumothorax has been wrongly diagnosed on account of the hyper-resonance with absence of breath-sounds. In these cases, the *bruit d'airain* cannot be obtained: and a skiagram may show the presence of aneurysm in the case of compression; or the lung retracted towards the spine in the case of pneumothorax.

If, on the other hand, the lung is collapsed as the result of the stenosis, the physical signs resemble those due to a partially absorbed pleuritic effusion, and the exploring syringe may be necessary for a final decision. A localised bronchiectasis should always suggest inquiry into possible obstruction as a cause. Where foreign bodies are in question, the history must, of course, be carefully considered. In suitable cases, especially when foreign bodies are concerned, the *bronchoscope* may be employed.

The **Prognosis** and **Treatment** are similar to those of obstructed trachea (*see* p. 481); tracheotomy is of course useless except for the removal of foreign bodies.

DISEASES OF THE LUNGS

EMPHYSEMA

The term emphysema (from ἐν, in, and φῦσα, wind) is rightly used to denote the extravasation of air into the subcutaneous or other tissues of the body (*surgical emphysema*), and into the interlobular or interstitial tissue of the lungs (*interstitial emphysema*). It is much less applicable to the disease of the lung now under consideration, for which, however, in medical parlance it is usually reserved. The alveoli of the lung naturally contain air; in this disease they are abnormally distended, and may be said to contain too much. So far the name emphysema (*vesicular emphysema*) may be justifiable; but the name *alveolar ectasis*, which has been suggested, is more correct.

Anatomy.—The essential change in emphysema of the lungs is a loss of elasticity, from weakening, and subsequent atrophy and destruction, of the elastic tissue contained in the alveolar septa. In consequence of this the walls of the air-vesicles yield to the pressure of the contained air, and become distended. This brings them into closer contact with neighbouring alveoli which are also dilating; and between the two the alveolar septum becomes atrophied. Soon a perforation is established through the septum between the two alveoli, then the whole septum is destroyed, and the two alveoli become one. In this process not only the elastic tissue, but also the whole network of pulmonary capillaries contained in the septum, disappears. If this is repeated extensively throughout the lungs, first, all the air-spaces are much enlarged, and in many places great blebs of lung-tissue simply containing air are formed; secondly, the elasticity of the lung necessary for expiration is reduced much below the normal; thirdly, the vascular area available for aerating the blood is greatly diminished; and fourthly, in most cases the lungs themselves are considerably enlarged. A lung affected with emphysema does not collapse when the chest is opened at the *post-mortem* examination, but even bulges out through the aperture. It is soft and inelastic, and yields to the pressure of the finger ("pitting"). In different parts of it, especially along the inner and lower edges, may be seen large blebs the size of peas or nuts; and the lung is unusually pale, and bloodless, and of a mottled gray colour. On section the larger blebs collapse; and the whole organ is much drier than usual, unless in some parts, such as the bases, which may have been the seat of a complicating bronchitis or œdema.

A variety (*small-lunged emphysema*) occurs in old people, as a senile atrophic change; the lung is not enlarged, and blebs are not numerous. The septa have atrophied so that alveoli have joined

together, and the lung is shrunken, inelastic, drier, and paler, and presents a less perfectly spongy structure than normal.

Ætiology and Method of Production.—No doubt many cases of emphysema result from bronchitis, and some from whooping-cough or asthma; but this will not account for all, as it is certainly common to find the indications of a slight emphysema in those who have never had any such illness. In all cases it is the failure or wearing out of the elastic tissue that is the essential lesion.

All elastic tissue tends to wear out, though not uniformly, with increasing years, as shown in the skin and elsewhere. Emphysema may, therefore, certainly develop quite spontaneously at different but generally advanced periods of life. It may be more directly induced by a number of laborious occupations which entail prolonged strain upon the lungs, the chest being held full of air for a long time, either to serve as a *point d'appui* for the use of the arms, or to supply air in a regulated way, as in playing upon wind instruments, glass-blowing, &c.; but it is sometimes found in people of early middle age whose circumstances are not in any way exceptional.

The mechanism in the case of glass-blowers and others is probably that the lungs are kept expanded during the regulated effort, or that the force of expiration is opposed by the obstruction in the work, and so the elastic tissue is kept unduly on the stretch. Prolonged coughing in bronchitis and whooping cough has the same effect; and in the former the secretions constitute an obstruction to the expiration, from which the elastic tissue necessarily suffers strain. The greater development of emphysema in certain parts of the lungs, especially the anterior margins and lower edges, and in the neighbourhood of old cicatrices at the apex, may be accounted for on Jenner's view—that when air is retained in the chest under great pressure, as when playing a wind instrument, or making any great muscular effort, it is the parts of the lung which are least supported by the surrounding structures which will be most subject to the air-pressure from within. These are precisely the anterior and lower edges in healthy lungs. When a portion of lung shrinks from chronic disease, the support which it afforded to the adjacent lobules is, of course, withdrawn; a local emphysema then arises, which is called *compensatory*.

Results of Emphysema.—These are of two kinds. In consequence of the loss of elasticity, expiration, which is largely effected by the spontaneous collapse of the lungs after inspiration, becomes more difficult; the lungs tend to increase in size; the chest enlarges in width and depth, assuming permanently the shape and position which are characteristic of full inspiration; the mobility of the chest is much diminished, since it ranges only between different degrees of inspiration, instead of between full inspiration and full expiration; the interchange of gases is less complete; and every attack of bronchitis is aggravated from the impairment of coughing power which follows on the above defects.

The other important factor is the *loss of capillary area*, and hence of aerating surface. From this results an obstruction to the pulmonary circulation, of a kind similar to that which is produced by disease of the left side of the heart. The tension in the pulmonary artery and right ventricle is increased, the right ventricle hypertrophies or dilates, or both, and the venous system becomes engorged, producing in course of time congestion and enlargement of the liver, œdema of the feet, legs, and trunk, and albuminuria.

Symptoms and Physical Signs.—The symptoms of emphysema are at first only shortness of breath; the cough and expectoration which are commonly present result from a co-existing bronchitis. The dyspnœa is especially seen on exertion in early stages, when the breathing is quickened and the patient readily pants; later on it may be always present, producing orthopnœa at night. In its worst forms the extraordinary muscles of respiration are in constant use; the clavicles are lifted; and the sterno-mastoids and scaleni stand out at each inspiration, striving to increase the tidal air; expiration is prolonged, laboured, and aided to their utmost by the muscles of the abdomen. The *physical signs* are characteristic. The chest is broad, deep antero-posteriorly, but short; it is often called barrel-shaped, from its enlargement, and from the increase of the antero-posterior diameter giving it rather a circular than a transversely oval shape. The shoulders are raised; the upper ribs are closer together, and the lower ribs wider apart than normal; and the epigastric angle is very obtuse, measuring 105° or more. The elevation of the ribs alters the relative positions of the nipple and the heart's impulse; the nipple is often found on the fifth rib, and the heart's impulse in the sixth space. But this last may be partly displaced by the enlarged lung. Percussion gives excessive resonance over the parts of the chest which are normally resonant, and an extension of the resonance over areas which are normally dull. Thus the hepatic and cardiac dulnesses are encroached upon, the right lung being resonant down to the sixth space or seventh rib, and the superficial heart-dulness being limited to the fifth cartilage and space below, or even disappearing altogether. Posteriorly, the resonance extends to its fullest limits downwards. On auscultation the inspiratory murmur is very much diminished or scarcely audible, and the expiratory murmur is much prolonged.

The enlargement of the lungs also affects the signs connected with other organs. The impulse of the heart is scarcely perceptible in the normal position, but epigastric pulsation is often present. The cardiac sounds are much less loud, from the lung overlying the heart. Any enlargement of the heart from dilatation or hypertrophy of the right ventricle may thus be concealed. The systolic murmur of tricuspid regurgitation is occasionally present at the lower end of the sternum. The liver and spleen may be slightly displaced downwards.

In small-lunged emphysema the chest is more nearly circular

in its outline, but it is not enlarged; the lungs do not cover the heart; and the heart is not hypertrophied, but atrophied. The percussion note is hyper-resonant, and the inspiratory murmur is feeble, but the expiration is not prolonged.

In both forms the rhonchi of bronchitis are frequently present; in extreme cases there are râles at the bases of the lungs, and the intercostal spaces are sucked in with inspiration.

Complications.—Chronic bronchitis is most frequently present, with or without bronchiectasis; and in late stages of the more common form, dilatation of the right ventricle, with anasarca and albuminuria. Bright's disease, with its cardio-vascular changes, may complicate it in old patients.

Diagnosis.—Its recognition depends upon the altered quality of resonance, and especially upon the extension of resonance over the præcordial area, and downwards over the liver. In the small-lunged variety the altered quality of resonance and the dyspnœa are the chief features. The Röntgen rays show a more extensive and lighter area over the lungs than in health; and a lower position and less extensive movements of the diaphragm.

Prognosis.—Actual recovery does not occur; only relief of symptoms. The duration of life depends upon the extent of the change, and the liability to bronchitis, or to cardiac dilatation. In most cases the final result does not come under several years.

Treatment.—This must be directed to improving the general health of the patient, to avoiding all risk of bronchitic complications, and to relieving these when they occur. Thus the patient should have nutritious and digestible diet, should be well clothed, live in warm, well-ventilated rooms, and avoid east winds and the night air. Tonics, such as cod-liver oil, iron, strychnia, and quinine, are of some value, since the disease partakes of the nature of degeneration. Attempts have been made to compensate for the loss of elastic tissue by different methods; thus Gerhardt advises assisting expiration by mechanical compression of the thorax; this is done by another person with the hands upon the lower part of the thorax for five or ten minutes every day. A patient of Strümpell's managed it himself by means of two boards, one on either side of the chest, which he brought together at each expiration. The compressed air bath, in which the patient breathes air at an additional pressure of two-thirds of an atmosphere, has been found useful at the Brompton Hospital.

For the accompanying bronchitis, expectorants such as ammonium carbonate in doses of 5 to 7 grains, vinum ipecacuanhæ, and the infusion or tincture of senega, should be given. Mustard plasters or linseed-meal poultices will afford some local relief. If the heart is failing, digitalis, strychnia, or other heart tonic must be used; and when anasarca is threatened, purgatives such as pulv. jalapæ comp., and diuretics, such as squill, acetate of potassium, spirits of nitrous ether, and citrate of caffein, should be given to relieve the overloaded venous circulation.

COLLAPSE OF THE LUNGS

(*Atelectasis Pulmonum*)

A distinction is often made between lungs that have never completely expanded (atelectasis) and those that have, after expansion, partly returned to the fœtal state (collapse). The former condition is congenital, and is seen in very weakly children, whose respiratory movements are insufficient to draw in the required amount of air. The latter results from conditions which prevent complete expansion of the lungs. These are (1) obstruction to the entrance of air, by the air-passages; (2) compression of the lung from without; and (3) paralysis of the diaphragm or intercostal muscles.

Obstruction may arise from chronic enlargement of the tonsils, adenoid growths in the naso-pharynx; much more often from the viscid, mucous, or purulent secretion of bronchitis, especially in children, and as a part of broncho-pneumonia; and in older people from constriction of the bronchus by cancer or by aneurysm or some other of the causes previously mentioned.

The causes of *compression* are numerous: in the chest itself it is most frequently due to pleural effusion, but also to enlargement of the heart, pericardial effusion, mediastinal tumours, aneurysms of the aorta, and angular curvature of the spine (kypho-scoliosis); in the abdomen, to the pressure of tumours growing from the upper surface of the liver, especially hydatids, abscess and cancer, of subdiaphragmatic abscesses, hydatid of the spleen, ascitic fluid, and ovarian tumours.

Paralysis of the diaphragm occurs in diphtheria, and other forms of multiple neuritis; and paralysis of the intercostal muscles in lesions of the upper dorsal portion of the spinal cord. The action of the diaphragm is no doubt hampered in many lesions of the lower thorax, or upper abdomen; and some of the results included under compression may be partly brought about by this factor.

The mechanism of compression is obvious; the air is forced out and the walls of the air-vesicles and bronchioles approximated. The explanation of collapse from obstruction is that when the to-and-fro movement of air in the lungs is prevented, the air imprisoned within the air-vesicles is absorbed, and the vesicles collapse. The operation of diaphragmatic (or intercostal) paralysis appears to be more rapid, and leads, according to Pasteur, to massive or lobar collapse, the lower lobes being involved in case of paralysis of the diaphragm, the upper lobes in intercostal paralysis. He ascribes it to the air being driven out directly by the elasticity of the lung, when left to itself by the failure of the inspiratory muscles.

Morbid Appearances.—Lung in a state of collapse or atelectasis has a violet or dark purple-gray colour, and is tough, airless, and dry on section. Isolated patches are seen to be slightly depressed below the general surface. Unless subsequently the seat of inflammation, they may be again expanded by forcible inflation with air.

Symptoms.—In congenital atelectasis the child is weakly, more or less livid, with rapid shallow breathing and feeble cry. With each inspiration the lower part of the chest is drawn in, and the intercostal spaces are depressed. Examination may elicit a little loss of resonance at the bases, and occasionally some râle, but feebleness of breath-sounds is the chief physical sign. The collapse of bronchitis is rarely extensive enough to reveal itself by auscultation, its distribution being lobular and scattered.

When collapse is more extensive and uniform different stages can be recognised by the physical signs. A very slight degree of collapse may occur at the bases from temporary disuse of the lung as a result of early pleurisy, or from prolonged dorsal decubitus. When the affected area of lung is auscultated, the breath-sound is very feeble; if the patient breathes deeply, there is a louder vesicular murmur, and at the end of it a fine dry rustling râle (crepitation), which is due to the fresh expansion of hitherto collapsed air-vesicles.

When the collapse is greater, as from pleuritic effusion, and yet not complete, the physical signs are dulness, diminished tactile vibration, and either diminished or faintly bronchial breath-sounds. This may also occur from pressure backwards of a large heart or aneurysm, or a distended pericardium. In complete collapse from compression or from obstruction of a main bronchus, there is entire absence of resonance, breath-sounds, vocal resonance, and tactile vibration—a negation of all the signs due to the presence of healthy lung. If the collapse is due to causes other than compression, the corresponding part of the chest may be retracted: and in either case there is supplementary breathing in some other part of the lungs—the other side if one lung is involved, the apices if the bases are collapsed, and *vice versâ*.

In the collapse due to diaphragmatic or intercostal paralysis immobility, dulness, and absence of breath-sounds, or sometimes bronchial breathing, are the physical signs.

The chief symptom is dyspnœa, in proportion to the amount of lung involved: even with much collapse the dyspnœa may be very little as long as the patient is at rest.

Treatment.—In most cases of collapse, the primary cause must be discovered and treated. In congenital atelectasis, the treatment must be supporting. The child should be kept warm in a well-ventilated apartment, the chest may be gently stimulated by friction, and proper feeding should be secured. In older children, bronchitis, rickets, or congenital syphilis must be met by appropriate treatment.

ŒDEMA OF THE LUNGS

This consists of the exudation of serous fluid into the interstices of the lung, and into the air-vesicles and smallest bronchi.

Ætiology.—It results from disturbances of the circulation, and from general blood diseases; and its most frequent causes are valvular disease of the heart, and acute and chronic Bright's disease. It is not infrequent in elderly people with failing hearts, as a permanent condition during the last few years of their lives; it forms part of the condition known as hypostatic congestion in prolonged febrile illnesses, such as enteric fever; and an inflammatory œdema generally accompanies acute pneumonic processes. Œdema may occur as an acute process in the course of chronic renal disease, or even of apparent health. Local œdema may result from the pressure of tumours or of aneurysm on the pulmonary vessels; and finally, in diseases involving the lung, like pneumonia or pleuritic effusion, œdema of the formerly healthy lung contributes to the fatal termination.

Symptoms.—The symptoms which are due to the œdema, in addition to those of the primary lesion, are dyspnœa, orthopnœa, more or less cyanosis in extreme cases, cough, and expectoration of abundant frothy serum, or sero-sanguineous fluid. The chest is resonant, or at most shows some slight impairment of the note at the bases behind; here the breath-sounds are deficient, and there are heard only abundant fine and medium râles. In the acute form above referred to (*acute suffocative œdema*) the patient is taken suddenly with dyspnœa, orthopnœa, sense of suffocation, cyanosis, small rapid pulse, and expectoration of large quantities of colourless, frothy, watery fluid. This may be quickly fatal, or subside in the course of a few hours. There may, however, be no expectoration until some days have elapsed; and, indeed, none at all, in some quickly fatal cases. In the final œdema of pneumonia, the râles are audible over the whole of the hitherto healthy lung.

Morbid Anatomy.—A lung affected with œdema is bulky, heavy, and exudes when incised an immense quantity of serous, slightly blood-stained fluid.

Treatment.—This is chiefly to be directed to the primary cause. Heart disease requires prompt treatment by the usual cardiac tonics, diuretics, and purgatives. In Bright's disease, diaphoretics and purgatives, or the vapour bath, should be employed. In elderly people with failing hearts, cardiac and general tonics are desirable. Ammonium carbonate is valuable, either by acting as a direct expectorant, or by stimulating ventricular contractions, and thus facilitating the circulation of blood through the lung. In the very acute cases venesection and nitrite of amyl or nitro-glycerine may be employed in early stages if arterial tension is high: but in later stages the heart's action must be supported by strychnia, digitalis,

ammonia, atropine, or alcohol. Atropine may also be useful in checking the pulmonary secretion.

PNEUMONIA

Inflammation of the substance of the lung, as opposed to the bronchial tubes, is called pneumonia. As an *acute* disease it leads to consolidation by exudation into the air-vesicles of inflammatory products, which are usually absorbed in the course of recovery. In a *chronic* form it causes a dense fibrous transformation of the interstitial tissue, which is permanent. Of acute pneumonia, two typical forms can be distinguished from one another by the following features: *Croupous* pneumonia occurs at all ages, but more often in adults, affects large portions of the lung at the same time, and is hence called *lobar*, and has all the characteristics of a specific infectious disease, with a limited duration, a quick recovery, and sometimes epidemic prevalence. *Catarrhal* pneumonia or *bronchopneumonia* affects chiefly infants, children, and elderly persons, invades several small areas of the lung, having a *lobular* distribution, and is much less definite in its course and modes of onset and termination. The two forms differ also in their histology and bacteriology. These distinctions are, however, by no means absolute, and some of the difficulties of recognition and distinction will be pointed out after the two have been separately described.

Chronic or *interstitial pneumonia* constantly forms a part of chronic pulmonary tuberculosis and of bronchiectasis, and only in a small number of cases stands alone as the primary lesion.

CROUPOUS PNEUMONIA

(Acute Lobar Pneumonia)

Ætiology.—The disease occurs in both sexes, but it is twice as common in males as it is in females, the difference between the two sexes being least marked in the very young, and in old people. It is seen also at all periods of life from infancy to old age, but it is more frequent in adults up to middle age. It occurs much more often in the winter and spring than in the summer and autumn; when the temperature is undergoing sudden changes, when the winds are east or north-east, or when the weather is wet or cold. Habits and occupations which involve exposure dispose to pneumonia, and it is probable that persons of weak health, or suffering from mental depression, or those who do not have sufficient nourishment, are more liable to it than the robust and strong. Intemperate habits also dispose to it, and greatly increase its mortality. An attack does not exempt from another: indeed, pneumonia is said to have occurred as many as fifteen or twenty times in the same patient, but more than two attacks are not very common.

Cold or chill often seems to be a determining event, but can act

only by favouring the invasion of the micro-organism which is the direct cause. Insanitary surroundings are also sometimes influential in the same way. Cases of direct contagion appear to be undoubted; and many instances are recorded in which pneumonia has spread rapidly through villages, large buildings, or households, precisely like an epidemic fever. Croupous pneumonia also occurs as a complication or sequela of some other diseases, and especially of mitral disease, acute nephritis, some infectious disorders, and diabetes.

Pneumonia is, indeed, a specific infectious disease, with the primary seat of infection in the lung; and the infecting organism is the diplococcus pneumoniæ of Fränkel, diplococcus lanceolatus, or *pneumococcus*, which is found in the lungs and sputum, and in severe cases in the blood. Other organisms are also found in small numbers—namely, Friedländer's bacillus, and streptococci and staphylococci. The pneumococcus appears to be widely diffused; it is found in the mouths of healthy persons, but is then of a virulence very much less than that which it possesses as a cause of pneumonia. It also occurs as a cause of inflammation in various other parts of the body, either as a secondary infection from the pneumonia, or as a primary infection through channels other than the lung. Thus there occur pneumococcal pleurisy, empyema, peritonitis, meningitis, arthritis, enteritis, endocarditis, pericarditis, nephritis, endometritis, and subcutaneous, intramuscular, or intravisceral abscesses; and in contrast with the usual change taking place in the lungs, many of these infections are from the first suppurative or pyogenic. The co-existence of two or more of them in the same patient constitutes *multiple pneumococcal infection*, or *pneumococcal septicæmia*.

Morbid Anatomy.—In pneumonia, the part of the lung affected is converted from a spongy structure into a solid mass. In the earliest, or first stage of *congestion* or *engorgement*, the lung is heavy, reddish-brown in colour, exudes a frothy, reddish serum on pressure, and breaks down more readily than in health. The capillaries are dilated and tortuous from distension with blood, and minute hæmorrhages may be present. In the second stage—called *red hepatisation*, from the resemblance which the consolidated lung bears to the liver—the organ is of a dull red colour, finely granular on section, completely airless, solid, sinking in water, but breaking down readily under the pressure of the finger. The contents of the alveoli, which may be detached in fine granular masses, are seen to consist of fibrin, containing red blood-corpuscles and a few leucocytes. The third stage, *gray hepatisation*, is also characterised by its solidity, but the colour is grayish-yellow or simply gray, and the surface is less granular than that of the red stage. Microscopically, it differs from the latter in that the air-cells and alveolar walls are crowded with leucocytes, while fibrinous exudation and red corpuscles are in very small quantity. The change of colour is attributable to the leucocytes in the alveolar walls, and to the decolorisation of formerly extravasated red corpuscles, but mainly to the blood in the vessels of the alveolar walls being prevented circulating by the pressure of

the infiltration. A fourth stage, that of *purulent infiltration*, is also described; but this is regarded by some as only an extreme condition of gray hepatisation. The lung is softer, yellowish in colour, and yields to scraping or pressure a quantity of yellow purulent fluid, which is provided by the disintegration of the infiltration filling the air-cells, the leucocytes becoming fatty and granular. A true *abscess*, however, is exceedingly rare as the result of typical acute pneumonia. It is doubtful whether the stage of purulent infiltration is ever reached in cases that recover; it is true, recovery, or *resolution*, is sometimes accompanied by physical signs (*redux crepitation*) which indicate that the exudation is softening into fluid. But many patients get well without such evidence, and with so little expectoration that the removal of the exudation can only be explained by its absorption, either directly by the lymphatics or by the agency of leucocytes; and in few cases is the amount of sputum very considerable. In the rare cases which do not recover, but are not quickly fatal, *gangrene* or *chronic pneumonia* or *bronchiectasis* may be the result.

The inflammation of the substance of the lung is accompanied, in a large proportion of cases, by inflammation of the pleura; this often causes pain at the commencement of an attack, may be recognised by friction sound, and may not be again evident during the course of the disease. Pleuritic lymph may be discovered after death, of which there was no evidence during life, and serum, or pus, may be formed in considerable quantity. The double lesion may be spoken of as *pleuro-pneumonia*, but the name is not generally used except for cases in which the pleurisy is clinically a prominent feature.

Localisation.—Pneumonia is nearly always partial, affecting the base more often than the apex, and the right lung somewhat more often than the left. Beginning at the base of one lung behind, it extends upwards to the apex, as well as forwards; or it may extend downwards from the apex; or commence in the centre and spread upwards and downwards. Its progress appears sometimes to be stayed, or checked for a time, at the line of the lobar fissures. Occasionally both lungs are affected, but the disease commonly begins in one earlier than the other.

Symptoms and Physical Signs.—Shortly stated, the symptoms of a typical pneumonia are pyrexia, beginning suddenly with rigor, continuing, with a temperature of 103° or 104°, for five to eight or more days, and ending suddenly or gradually; and pain in the side, dyspnœa, cough, expectoration of viscid sputum stained with hæmoglobin. The physical signs are those which indicate consolidation of the lung—viz., dulness, bronchial breathing, bronchophony, and increased tactile vibration.

First Stage.—A rigor occurs, in a large proportion of the cases, as the first definite sign of illness; the temperature rises to 102°, 103°, or 104°, and there is well-marked pyrexia, with the usual loss of appetite, furred tongue, and malaise. The symptoms may be at first vague, accompanied perhaps with pain in the head or back; or

the implication of the lung may be indicated by shortness of breath and severe pain in the side, attributable to pleurisy. Auscultation at this early period may detect nothing, but sometimes there is heard a fine dry crepitation, which has been compared to the noise produced by rubbing between the finger and thumb a lock of hair near the ear; it is mostly heard towards the end of a deep breath, but sometimes during the whole of inspiration; and it is explained by the separation of the walls of the alveoli rendered unnaturally adhesive. More often the first deviation from the normal is a marked diminution or loss of the vesicular murmur, over the area which subsequently gives the signs of the second stage, or consolidation. The percussion note may be still unaltered, or only slightly less resonant than normal.

Even as early as this there may be slight cough, with the characteristic *rusty sputum*. This is brought up as a mass of transparent, airless, jelly-like mucus, of a yellow, orange, russet-brown, or even bright red colour, and extremely viscid, so that it adheres to the side or bottom of the vessel with little or no tendency to flow. The pneumococcus can be detected in the sputum by Gram's method of staining.

The physical signs of the *second stage*, or stage of consolidation, are often rapidly developed. There is decided dulness over the part of the lung affected. Over the same area there is bronchial breathing, at first perhaps soft and distant, but in a short time, loud, ringing, and metallic. If the patient speaks there is loud bronchophony, the words uttered being often distinctly heard, and apparently shouted up into the stethoscope; whispered words are also distinctly transmitted. The fine crepitation heard as an early sign may still be audible in portions of lung which are being involved by the spreading inflammation; but over areas, which give loud bronchial breathing and bronchophony, crepitations may be heard, somewhat coarser, and generally markedly consonating. Tactile vibration is often, but not always, increased. Rarely, instead of dulness, there is a peculiar tympanitic note, or even a cracked-pot sound; and these have been attributed to the presence of a thin layer of still spongy lung between the hepatised portion and the surface. During this development of the physical signs the patient is almost necessarily confined to his bed, but he is often obliged to have the shoulders raised; his cheeks and forehead are flushed, with perhaps a slight tinge of jaundice; his eyes are bright, and show a vivid consciousness of his distress; his breathing is quick and shallow rather than laboured, and the respiration may rise to 40, 50, 70, or even 80 in the minute. The pulse is quickened, but not in proportion to the respiration; it may be 100 to 120, or somewhat higher; thus the pulse respiration ratio is altered from the normal 3 : 1 or 4 : 1 to 2 : 1 or $1\frac{1}{2}$: 1. The temperature is maintained generally at a high level, 103° to 105°, with little variation; and the skin is dry, and gives a sense of pungent heat to the hand placed on it. The cough, which is usually, though not always,

present, is not very frequent, is hard, dry, and often painful; and the viscid, rusty sputum is brought up with difficulty. The urine is scanty, high-coloured, acid, and deposits urates; the chlorides are much diminished and may be absent, and there is not infrequently a small quantity of albumin. There is generally leucocytosis

FIG. 46

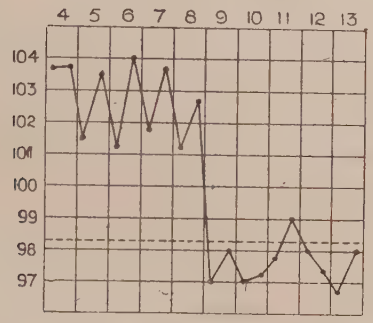


Chart of a Case of Pneumonia, with Crisis on the Eighth Day.

with increase in the polymorphonuclear cells, which persists for a long time in severe cases. The patient often retains his consciousness entirely, or may wander a little at night.

The general condition of the patient continues very much the same for some days, or more often there is an increase in the severity of the symptoms. The pulse and respiration are quicker, the temperature continues high, the tongue becomes drier and browner, and the delirium at night is more decided. The physical signs are generally observed to alter from day to day, indicating the spread of the consolidating process, so that crepitation and bronchial breathing extend higher and higher up the chest, until the apex is involved, and the physical signs may be apparent in front under the clavicle.

Later Stages.—One cannot distinguish, clinically, the stage of gray from that of red hepatisation. The stage of *resolution* can be recognised by many indications.

When the illness is apparently at its worst, improvement takes place, in many cases quite suddenly. On the sixth, seventh, or eighth day, in a large proportion of cases, the temperature, the pulse, and respiration fall, in the course of twelve or eighteen hours, nearly to their normal limits; the tongue becomes moist; and the patient feels himself in all respects better. This *crisis* may be accompanied with profuse sweating, or with diarrhœa. In more than half the cases the fever ends more gradually (*lysis*), occupying from four to five days while falling from the acme to normal.

In either case the pulse and respiration fall with the temperature, the physical signs quickly or more slowly clear up, the bronchial breathing becomes fainter, and the dulness less marked. In this

stage there is often heard a well-marked crepitation, louder, coarser, and moister than the early pneumonic sound; it is called *redux crepitation.* The sputum also changes, losing its characteristic tinge, becoming yellow or green, muco-purulent, and at the same time less viscid.

In fatal cases, death occurs from failure of the heart, or from œdema of the hitherto unaffected lung, or from both combined. All the symptoms are aggravated—the respirations are increased in frequency; the pulse is quick, small, and feeble; the face becomes cyanosed; the physical signs of dilatation of the right ventricle may be observed; the tongue is dry, brown, and cracked; delirium is more or less continuous, and muttering and coma gradually supervene. On auscultation, loud, coarse râles are heard on both sides of the chest. As the patient becomes feebler, the temperature falls, the skin becomes cold and is bathed in profuse perspiration. Death commonly takes place during the height of the illness, between the fifth and the tenth days. Occasionally, however, a pneumonia runs a fatal course in two or three days.

Complications and Sequelæ.—The former are mostly the result of secondary pneumococcal infections. *Pleurisy,* with formation of lymph or serum, is the most frequent. *Empyema* is not so common, but it should be suspected if fever continues into the third week, with dull percussion note, and disappearance or change of the bronchial breath-sounds. *Pericarditis* is frequently associated with empyema on the left side. *Peripheral neuritis, nephritis, peritonitis, suppurative meningitis,* and *arthritis* are among the rarer pneumococcal complications. Rarely, a true *pneumococcal pyæmia* has occurred with suppurative arthritis and pustules and abscesses under the skin, yielding a thick greenish pus containing pneumococci in pure culture. *Malignant endocarditis* (especially of the aortic valve) has been rather often seen in association with pneumonia, and as a result of its specific organisms. In a small number of cases there is pronounced *jaundice;* a faint icteric tinge is more common. The cause of jaundice in pneumonia is not clear; it is not due simply to extension, since it occurs both in apical and in left-side pneumonia. Acute *dilatation of the stomach* sometimes occurs in the course of the illness, and *parotitis* may ensue in severe cases. *Chronic pneumonia, gangrene* and *abscess of the lung,* and *bronchiectasis* are rare sequelæ.

Diagnosis.—In the early stages of rigor and high fever pneumonia may be indistinguishable from other *acute illnesses,* such as typhoid, scarlatina, or small-pox. Frequently the pain or distress in one side of the chest will indicate acute disease there, and the absence of breath-sounds, or the fine crepitations, at one spot, followed by dulness, bronchial breathing, and bronchophony, will show the nature of the illness. But the pain may be very misleading; it may be so low in the back as to suggest variola; and it frequently extends to the abdomen, or is felt chiefly in the abdomen, so that *appendicitis, peritonitis,* or *cholecystitis* may be first

thought of. A careful watch on the pulmonary bases is required to guard against error. In other cases a short cough, with expectoration of rusty sputum, will occur before the development of the physical signs. These last may, indeed, be delayed for five or six, or even ten days, and they may require much looking for and be first found in unlikely places, such as over the scapula, or at the top of the axilla. The absence of rashes characteristic of the exanthemata, the rapidity of respiration out of proportion to the pulse, the flushed face and bright eye, the characteristic sputum, and the presence of herpes about the mouth are useful points in making a diagnosis. An examination of the blood may help, as the presence of leucocytosis excludes typhoid fever, malaria, and influenza. The Röntgen rays are also of value, as the consolidated lung casts a definite shadow even in cases where the affected lung is so remote from the surface as to yield no physical signs. The movements of the diaphragm on the same side are limited, and the right side of the heart is often seen to be enlarged.

When physical signs appear, it has to be determined whether pneumonia or *pleuritic effusion* is present, or a combination of both. The diagnosis of these two conditions from one another will be dealt with under Pleurisy; it will be sufficient to say here that pleuritic effusion, though often accompanied by bronchial breathing, causes more absolute dulness than pneumonia, and weakens or abolishes tactile vibration. When they co-exist the pneumonia is often masked by the physical signs of the pleuritic effusion which lies over it, whereas the pneumonia may be signalised by the rusty sputum, and the pyrexial conditions, which are commonly more pronounced than those of pleurisy. As pneumonia very rarely becomes chronic, physical signs of consolidation persisting for weeks with continued pyrexia are almost always due to pus or serum in the pleural cavity. The diagnosis from broncho-pneumonia is considered later (*see* p. 517).

Prognosis.—The mortality of acute lobar pneumonia is about 17 per cent. The disease is more fatal to the intemperate, and to those who have been insufficiently fed. Apart from these considerations, it is difficult at the onset of a case to say what the end will be. Early or violent delirium, failing pulse, cyanosis, the rapid implication of the whole of one lung, the spread of the disease to, or the occurrence of œdema in, the other lung, are all symptoms of bad augury.

Treatment.—The patient of necessity takes to his bed, and generally in the height of the disease requires to be supported in a semi-recumbent position by means of pillows or bed-rest. He should have abundance of fresh air, in a freely ventilated room, no less than in any other infectious disease. The diet should consist of milk and beef-tea, or mutton broth, administered in small quantities, frequently. It is almost certain that no drug has any direct influence upon the inflammatory process as such, though much assistance may be obtained by influencing the resisting processes, and supporting the heart in severe cases. Treatment on

bacteriological lines has been disappointing: in acute pneumonia neither immune sera nor bacterial vaccines have been of much value; but in chronic suppurative affections due to the pneumococcus, while sera are useless, vaccines, especially those prepared from an organism isolated from the case concerned, have produced good results.

In early stages the bowels should be opened, and a free action of the skin should be encouraged by the use of acetate or citrate of ammonium, with small doses of Dover's powder. This last will relieve the pleuritic pain, or opium may be more frequently given in small doses (3 to 5 minims of tincture) with the saline, or a few leeches may be applied. The application of linseed-meal poultices, of linseed meal sprinkled with mustard, and of hot flannels wrung out of turpentine gives temporary relief, but the influence upon the disease is doubtful. On the other hand, ice applications (ice-bags, or pieces of ice between layers of flannel) relieve the sense of tension, tend to reduce temperature, and are liked by the patients. In mild cases this may be all that is required, but in the severer cases delirium and increasing prostration will have to be met. For the former chloral, chloralamide, and potassium bromide may be employed; but when there is much dyspnœa chloral must be given with caution, because of its depressing effect upon the heart and respiration. For the same reason, morphia must be sparingly used in the later stages. The subcutaneous injection of hyoscine hydrobromide ($\frac{1}{100}$ gr.) is often useful and safer. For the increasing cardiac failure which constantly accompanies delirium, and which is the main cause of a fatal end, probably the injection of liquor strychninæ in doses of 2 to 5 min. every six hours is the safest and most efficient treatment. Digitalis may also be given in these circumstances; and small quantities of brandy or other spirit, up to the extent of three or four ounces daily. Calcium salts (chloride, 10 grains every four hours) have also been advocated as cardiac stimulants. Cases in which it is apparent from much cyanosis and dyspnœa, commencing râles in the opposite lung, and from percussion of the cardiac region to the right of the sternum, that the right ventricle is dilated, may be relieved by leeches over the præcordia, or may require venesection. Inhalations of oxygen are sometimes of great value, and ammonium carbonate (5 to 7 grains every three or four hours), especially when there is much secretion in the tubes. When the crisis is past, and the temperature has fallen to the normal, the treatment requires simply to be directed to the strengthening of the patient by the administration of quinine and other tonics, since the sequelæ of acute pneumonia are few and infrequent.

BRONCHO-PNEUMONIA

(*Catarrhal Pneumonia. Lobular Pneumonia*)

Ætiology.—This form of pneumonia occurs most often in children under three years of age. It often results from the extension of bronchitis to the air-vesicles; it is a frequent complication of measles and whooping-cough; and also occurs after other infectious diseases (diphtheria, scarlet fever, and influenza). Ill-nourished children and those habitually breathing an impure air are thought to be more liable to broncho-pneumonia, and it is probable that rickets, by weakening the thoracic movements, also disposes to it. Lobular pneumonia occurs in adults from inhalation of foreign particles, especially septic materials from the throat, into the lungs (*inhalation pneumonia*); and it results from metastasis in septic diseases, like pyæmia.

The micro-organisms found in broncho-pneumonia are: the pneumococcus, streptococcus pyogenes, staphylococcus albus and aureus, and Friedländer's bacillus pneumoniæ. Streptococci are more frequent and pneumococci less frequent than in croupous pneumonia, and the former are more frequent in the cases which are secondary to bronchitis.

Morbid Anatomy.—In broncho-pneumonia the consolidation is scattered throughout the lung in the form of nodules, mostly separate, but sometimes aggregated closely together, so as to form larger masses (*confluent broncho-pneumonia*), but even then still to be distinguished by the eye from each other. On section they are seen as patches, more or less rounded in form, about the size of a pea, reddish-brown, gray, or grayish-yellow, with a less marked granular appearance than in ordinary pneumonia, slightly raised above the surface, solid to the touch, yet soft, friable, and yielding some corpuscular fluid on squeezing. Under the microscope, the contents of the alveoli are seen to be mostly large cells, of an epithelial type, the result of proliferation of the cells lining the alveoli; but inflammatory products from the finer tubes can also be recognised, which have been sucked into the alveoli by the inspiratory efforts. Patches of collapsed lung accompany the consolidation; and when the lobules near the surface are consolidated, they generally give rise to some pleurisy.

Symptoms and Physical Signs.—The former are cough, dyspnœa, and pyrexia; the latter vary with the extent and position of the separate lesions. If the child has already a cough, with rhonchi and râles over the chest, from a preceding bronchitis, the implication of the alveoli may be indicated by a rise of temperature to 102° or 103°, by the cough becoming short, dry, and painful, and by the râles becoming more abundant, and taking on a consonating character. But in many cases there are no rhonchi, and the physical signs consist of one or more areas, more or less extensive, in one or both lungs, in which rather sharp crackling râles are

heard, with little if any change in the percussion note; or areas, also irregular in distribution, over which there is dulness, with bronchial breath-sounds and bronchophony, due to the aggregation of a sufficient number of consolidated lobules. Such areas may enlarge or diminish, and spread or clear up as the disease pro-

FIG. 47

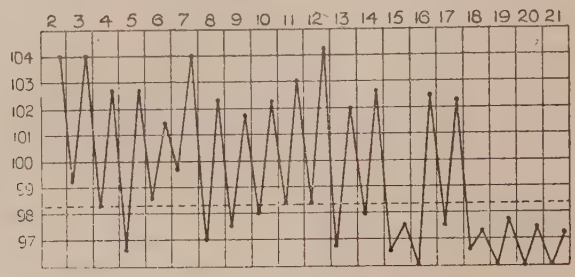


Chart of a Case of Broncho-Pneumonia.

gresses. The sputum consists of mucus with or without streaks of blood, but young children usually swallow it. Exceptionally there may be free hæmoptysis.

The course of the disease is not so definite as in croupous pneumonia. It may end in a week, but often goes on for three or four weeks, or even more. The temperature is generally remittent, or even intermittent, with oscillations of 3, 4, or 5 degrees between morning and evening (*see* Fig. 47); and the pyrexia, whether of short or long duration, may end abruptly as there shown, or fall more slowly by lysis. The breathing is rapid and shallow, and is effected in a manner which is often valuable in diagnosis. It has been called *pneumonic breathing*, though it occurs also in pleural effusion: there is a quick inspiration, the breath is held for half a second, expiration then occurs with a grunt, and inspiration again follows without an interval. The lower intercostal spaces are depressed during inspiration. There is much cough; the face is flushed, or in severer cases pale and livid. The pulse is quick and small. Delirium is often present. The physical signs frequently alter in the course of the illness, indicating the clearing up of disease at one part, and fresh outbreaks in others; and the disease often attacks both lungs. Recovery is mostly gradual, and not sudden, as in croupous pneumonia. Convulsions may precede death.

Sometimes, however, and especially in older children, broncho-pneumonia, with a lobular origin and catarrhal character of the *post-mortem* lesions, is clinically almost identical with the acute pneumonia of adults: it begins suddenly, runs a short course, terminates in crisis, and the physical signs indicate consolidation of a single extensive area of lung. Often in these cases the fever is of the intermittent type shown in Fig. 47, during the short time that it lasts.

Many cases of pneumonia following influenza are noted for the

absence of bronchial breathing, and the predominance of fine crackling râles, great variety in the duration of the pyrexia, but a general resemblance to croupous pneumonia in the other symptoms. In broncho-pneumonia, septic in origin, or secondary to local diseases, there is much variety in the physical signs and course of temperature.

Diagnosis.—Broncho-pneumonia may be confounded in its early stages with other *acute illnesses*, characterised by high fever, such as typhoid fever; and the liability of children to marked cerebral symptoms, from any acute illness, may lead to a diagnosis of *meningitis*. The preceding bronchitis, and the greater predominance of the chest symptoms, may assist; but an opinion may have to be suspended for a few days. Long-continued broncho-pneumonia may give rise to a suspicion of *tuberculosis*, in which high fever, universally scattered râles, lividity, and cough are prominent symptoms. In *capillary bronchitis* there are dyspnœa, lividity, and râles, but the râles are often confined to the bases; there is no bronchial breathing, and expectoration, if present, is purulent. The diagnosis from *acute lobar* or *croupous pneumonia* is often difficult in children; and conflicting statements are made as to the infantile ages at which croupous pneumonia and broncho-pneumonia are respectively more common and more fatal. When the consolidation affects a large area continuously on one side only, when the illness begins abruptly, has a temperature without much remission, and ends with crisis, it is commonly regarded as croupous. But confluent broncho-pneumonia may have the same situation and extent as a lobar pneumonia, and it is stated that a pneumococcal pneumonia—that is, a croupous pneumonia—takes on a lobular distribution in children (West). Bacteriological examinations are rarely possible in children, for they supply no sputa. Hence the course and duration of the fever remain as the chief guides. The two forms even co-exist in some cases.

Prognosis.—Though this form of pneumonia is much more fatal than the croupous variety, the prognosis in any given case must depend upon the general progress of the symptoms. Cases that are apparently desperate often recover, and an unfavourable opinion should be given with some caution. In the broncho-pneumonia of old people, and in that which is due to the inhalation of solid particles, the prognosis is more grave.

Treatment.—The treatment may be conducted on the same general principles as in the case of ordinary pneumonia. The room should be well ventilated, with free access of air to the patient; hot poultices are not now generally approved, and, on the other hand, very good results have been obtained with ice applications to the chest, even in quite young children. Expectorants, such as ammonium carbonate, one grain, and vinum ipecacuanhæ, two minims, should be given every four hours. Severe cases generally require stimulants to be administered rather freely—*e.g.*, for a child three or four years old, 20 minims of brandy every hour; or one or two minims of liquor strychninæ may be injected two or three times daily at this age, and smaller quantities in infants.

ABSCESS OF THE LUNG

Apart from the suppurating cavities determined by tubercular disease (*see* Phthisis) or bronchiectasis, abscess of the lung is comparatively rare. It has already been mentioned as a result of pyæmia, and of acute pneumonia. It may be determined also by foreign bodies entering the bronchi; and new growths, such as cancer, and probably syphilitic gumma, occasionally suppurate. As it is always a secondary occurrence, the symptoms of septic infection which it produces are added to or confounded with those of the preceding condition. The physical signs are not likely to be distinguishable from consolidation, until the abscess has burst, and discharged pus; and this may occur suddenly. The usual signs of a cavity, such as tympanitic resonance, cavernous or amphoric breathing, metallic râles, and pectoriloquy may then be heard. The multiple small abscesses of pyæmia are not recognisable as cavities: indeed, their presence is usually masked by the surrounding consolidation, or pleuritic effusion; and death may have taken place almost before the infarcts have softened down into fluid pus.

Treatment.—An abscess after acute pneumonia has occasionally been assisted by surgery; otherwise the patient must be supported by nourishing food, quinine, iron, &c.

CHRONIC PNEUMONIA

(*Fibrosis of the Lung*)

Ætiology.—This form of lung disease is comparatively rare, the great majority of chronic inflammations of the lung-tissue being associated with tubercle, and included under the term phthisis. The cases in which chronic pneumonia is independent of tubercle, and to which the names chronic pneumonia and cirrhosis or fibrosis of the lung are given, arise only in a few instances from a preceding acute croupous pneumonia; but catarrhal pneumonia is a more frequent antecedent. Chronic bronchitis, bronchiectasis, and chronic dry pleurisy appear to be the causes in other instances.

An important class of cases, in which chronic pneumonia succeeds to bronchitis, is that occurring in various manufactories, and known as *pneumokoniosis*. Here the repeated inhalation of an atmosphere laden with the dust of coal, metal, stone, cotton-fibre, fluff, &c., provides a life-long source of irritation. The disease has received different names according to the particular irritant concerned—*anthracosis* (coal dust), *siderosis* (steel), *silicosis* (stone). These cases are often the subjects of secondary tubercular infection.

Morbid Anatomy.—The characteristic feature of the lung affected with chronic pneumonia is the excessive development of fibrous tissue in its substance. At first the lung is traversed with bands of fibrous tissue, arising in the interlobular septa; in late stages the whole lung may be converted into a dense mass of fibrous

tissue, of various shades of gray, from the presence of pigment, tough in consistence, and creaking under the knife. In cases of dust disease, the lung is coloured black, red, or gray, according to the nature and quantity of the particles inhaled. With the growth of the fibrous tissue contraction takes place, and the lung may be reduced to two-thirds or half its natural size; the excavations commonly occur partly from dilatation of bronchial tubes, and partly from ulcerative processes in the lung-substance. Nearly all cases are accompanied by a chronic pleurisy, and the lung is fixed to the chest by a thick fibrous layer. The contraction of the lung leads to displacement of organs, and, as usually only one side is affected, the mediastinum is pulled in that direction.

Symptoms and Course.—The disease is generally chronic, and patients in whom it is recognised have generally complained for some months or years; but that the formation of fibrous tissue may begin very early is shown by a case in which the alveolar exudation was being organised into connective tissue at the end of three weeks (Kidd). The patients are short of breath, and have cough and expectoration, which vary with the extent of the cavities in the lungs. When there are large cavities, or much-dilated tubes, the cough may be paroxysmal, with abundant and perhaps fœtid expectoration. The patient is often thin, but may be well nourished, and is at any rate for a time free from the fever, night-sweating, and general constitutional disturbance observed in tubercular phthisis. Hæmoptysis is, however, often present. Some of the local conditions have been already indicated. The disease is generally unilateral, the corresponding side of the chest is retracted, the shoulder depressed, and the angle of the scapula tilted outwards; the impulse of the heart is shifted towards the affected side, and the healthy side of the chest is hyper-resonant. The affected side expands but little; it is dull on percussion. The respiratory sounds are feeble or distant; and if large cavities or much-dilated tubes (bronchiectasis) are present, the breathing may be hollow or tubular, with metallic or bubbling râles. Such cavities are more often situate about the middle level of the lung than at the summit, as in phthisis. Tactile vocal fremitus is commonly diminished. Thickening or *clubbing* of the finger-ends (*see* p. 531) is often pronounced. At first the compensatory action of the healthy lung suffices to maintain efficient oxygenation of the blood, but in time the right side of the heart becomes dilated, and cyanosis and dropsy ensue.

Diagnosis.—The condition has to be distinguished from ordinary phthisis, from chronic pleurisy with effusion, and from malignant growth in the chest. From *phthisis* the absence of fever and constitutional disturbance is the chief distinguishing feature; the disease is often rigidly unilateral, whereas phthisis rarely reaches an advanced stage in one lung without affecting the other; and tubercle-bacilli are not found in the sputum. *Pleuritic effusion* of old standing with retracted chest may closely resemble the fibroid lung, and exploration with a needle may be required to clear up the diagnosis.

Pleuritic fluid, whether simple or purulent, is generally accompanied by feverishness, and, on the other hand, crepitation and râles will be much in favour of chronic pneumonia. *Intrathoracic cancer* is likely to be associated with cachectic appearance, with irregular retraction of the chest, with pains and signs of pressure, with extensive consolidation, and with extension of dulness beyond the normal limits of the lungs; but one or more of these indications may be absent.

Prognosis is ultimately bad, but the course may be very slow, extending over ten or fifteen years. Death may take place from failure of the right heart, or from the gradually increasing exhaustion which follows profuse discharge.

Treatment.—The patient should be placed under the best possible climatic and hygienic conditions. He should have bracing air in the summer, but a warm climate in the winter; avoid exposure to chills at all times; and have nourishing diet and tonics, such as quinine, iron, and cod-liver oil. Cough, expectoration, and other symptoms should be treated as they arise, in the same manner as directed under Phthisis and Bronchiectasis.

GANGRENE OF THE LUNG

This is a comparatively rare disorder, which may arise, however, in a variety of circumstances. It is one of the terminations of acute croupous pneumonia, and occurs sometimes in phthisis; it may result from the invasion of the lung by adjacent diseases like cancer of the œsophagus, abscesses, and suppurating hydatid cysts, and from the pressure of aneurysm on the root of the lung; as a result of foreign bodies lodged in the bronchus, and from the presence of secretions retained in dilated tubes; from the inhalation into the lung of particles from septic diseases in the mouth, throat, larynx, or œsophagus, such as cancer of the tongue or larynx, sloughing of the tonsils, diphtheria, or cancer of the œsophagus; from particles of food drawn into the lung by accident, or during vomiting, especially in persons who are drunk, insane, comatose, or suffering from laryngeal paralysis, or from impure water inhaled during immersion. Gangrene of the lung is also sometimes caused by septic particles brought to it by the blood-vessels, as in various pyæmic processes, after otitis, bed-sores, puerperal disorders, &c. Practically all these causes are frequently in operation, without causing gangrene; this is more likely to occur under the influence of certain predisposing conditions—namely, marasmus, old age, intemperance, diabetes, general infectious diseases in their typhoid and adynamic stages, and some paralytic and mental disorders. The micro-organisms concerned are usually staphylococcus pyogenes aureus, and albus.

Morbid Anatomy.—The affected portion of lung is of a dirty, greenish-brown, or black colour, soft, readily breaking down, or even diffluent; and often emitting an offensive odour. It is gene-

rally surrounded by consolidated pneumonic tissue, into which it may gradually pass, or from which it may be more or less sharply marked off by a line of demarcation; thus in some cases the lesion is diffuse, in others definitely circumscribed. The gangrenous tissue may break down, and be expectorated, so as to leave a cavity with ragged, shreddy walls; and occasionally such a cavity opens into the pleural sac and causes pyo-pneumothorax.

Symptoms.—Gangrene of the lung occurring, as it often does, as a secondary lesion, just before death, may be readily overlooked. On the other hand, its symptoms may stand alone, or overshadow those of the primary lesion. Fœtid expectoration and fœtid odour of the breath are the most prominent. The latter may be very penetrating; it is carried to a great distance, and makes it almost impossible for other persons to live in the same room with the patient. The sputum is dirty gray, or greenish-brown, or black, from altered blood; and either fragments of gangrenous lung-tissue are found, or the microscope detects the typical elastic fibres (*see* p. 534). Occasionally hæmoptysis takes place. Cough, pain in the side, and irregular, and mostly intermittent, pyrexia are also present. The physical signs are those of consolidation and cavity proportionate to the extent of lung diseased—viz., dulness, bronchial or cavernous breathing, bronchophony, and medium or coarse râles; but their value in diagnosis must depend a good deal on the preceding disease, if any. The illness may begin with rigor and pain in the side, or with hæmoptysis, or with recurring attacks of fever and fœtid expectoration; in most cases these are soon followed by prostration, with quick small pulse, dry tongue, and death at no great distance of time. Some cases, however, last for months or years with much variation in the intensity of the symptoms, but without escaping a fatal termination. And in a few cases, with probably a very small patch of gangrene, recovery actually takes place.

Treatment.—This is similar to that of fœtid bronchitis. Antiseptic inhalations (creosote, carbolic acid, menthol, eucalyptus oil, thymol), or the antiseptic respirator should be frequently used. Guaiacol (3 to 5 minims) and oil of turpentine (10 to 16 minims) may be given internally, and the strength should be supported by quinine, cinchona, iron, ammonia, good food, and sufficient stimulants. A gangrenous cavity may sometimes be amenable to the surgical treatment of antiseptic incision and drainage.

PHTHISIS

(Pulmonary Tuberculosis. Consumption)

Tuberculosis of the lung occurs in two forms. In one there is a general distribution of minute tubercles throughout the organ, arising acutely, and determined presumably by the carriage of the tubercle bacilli by the blood or lymph-vessels, either from some

other part such as a bronchial or cervical gland, or a joint or kidney; or less commonly from a focus of chronic disease in the lung itself. This acute form often forms part of a general miliary tuberculosis (*see* p. 154). In the other form the tubercle bacilli are deposited, possibly from the air-passages, and multiply in one small part of the lung, usually the apex, and spread with very varying degrees of rapidity to other parts of the lung by means either of the bronchial tubes, or of the connective tissues. This constitutes *phthisis* (*φθίω*, I waste), or *pulmonary consumption*. It is thus at first entirely local.

The essential features of the disease are the formation of *tubercles* as the result of the irritation of the bacilli; the occurrence of pneumonic processes in connection with the tubercles; the consolidation of portions of the lung; the subsequent breaking down of tubercular and pneumonic areas into cavities; suppuration and the discharge of *débris* of lung-tissue by expectoration; and general constitutional disturbance of varying severity. The later changes are assisted by the action of other organisms, especially the pneumococcus and the staphylococcus pyogenes. The complications and associated lesions are explained by the spread of the process in the lungs, or by the co-existence of the tubercular lesions in other organs.

Ætiology.—The ætiology of tuberculosis has been already discussed (*see* p. 151); and here need only be considered if its appearance in the lungs rather than elsewhere is determined by special factors. It has been already stated that pulmonary tuberculosis is most common in young adults: in many it is preceded by no ascertainable lesion; in many the patient has been ill-nourished or anæmic for some months or years before the invasion; and it much less commonly attacks those who are well nourished and above the average weight.

Sometimes phthisis is determined by a recent acute illness such as typhoid fever, influenza, bronchitis, measles, or whooping-cough; sometimes at a longer interval by pleurisy with or without effusion, which may appear to have recovered completely; or by a local tuberculous lesion such as a suppurating cervical gland, a diseased joint, or scrofulous dermatitis.

Morbid Anatomy.—Tubercles form and develop in the lungs in the most typical way, with their adenoid structure, giant-cells, and bacilli, and their tendency to caseate and break down (*see* p. 150). They occur first in the interstitial tissues, especially in the alveolar walls, and in the peribronchial, perivascular, and subpleural tissues. From the alveolar wall they invade the air-vesicles, and may seem afterwards to be situate in them; and the tubercles situate in the walls of the small bronchi will project into and narrow their calibre. This simple growth of tubercle constitutes the *first stage* of phthisis.

After a time the tubercles are succeeded by pneumonic processes, of catarrhal and, less often, croupous kind, by which small areas of consolidation are produced, which may be more or less

intermixed with masses of tubercles now becoming altered by caseation from the gray to the yellow variety. This is the *second stage*, or stage of *consolidation*.

The *third stage* is that of *excavation*, or the formation of *cavities* or *vomicæ*. It arises by the breaking down and disintegration of cheesy tubercles, and of pneumonic lung. By a mixed process of caseation and suppuration the cavities become larger and larger; adjacent cavities run into one another, and ultimately the lung may be extensively hollowed out. In their earlier stages the walls are often formed of caseous deposit; but in old vomicæ they are quite smooth, like mucous membrane. They are often traversed by bands, or trabeculæ, which contain pulmonary vessels. The vessels resist the destructive process; whereas the bronchi are generally ulcerated in proportion as the cavities enlarge, and into each cavity one or more bronchi open, often by an aperture much narrower than the calibre of the tube above. The contents of vomicæ are caseous matter, *débris* of lung-tissue, and pus. The latter predominates in the older cavities; the quantity is very variable, and it may be so small, under certain circumstances, that no expectoration takes place for considerable periods. It is only rarely that decided putrefaction takes place in phthisical cavities.

But in the majority of cases this process of destruction does not have full play. The inflammatory changes present varying changes of activity in different cases; and the mischief may be stopped for long periods one or more times in its course, or may even become abortive at an early date, and go no farther. The development of *fibrous, connective,* or *cicatricial* tissue is the important agent here. It is rarely absent in any but the acutest cases, and in the chronic cases it forms a large proportion of the remaining tissue of the diseased lung. In the consolidated lung there are numerous bands running in the course of the interlobular septa, surrounding the bronchi, the blood-vessels, and the cavities, and forming a dense layer under the visceral pleura. The fibrous tissue is frequently deeply pigmented, and is mixed here and there with caseous masses. By its contraction it tends to diminish the size of the cavities, and opposes some resistance to destructive processes; and in some favourable cases a small deposit of tubercle may be ultimately converted entirely into a mass of pigmented fibrous tissue, which, indeed, replaces a similar amount of healthy lung, but is otherwise harmless. In these cicatrices it is not uncommon to find calcareous particles, from the deposit of calcium salts in the caseous material; and around such a cicatrix may arise the condition known as *compensatory emphysema* (*see* p. 501).

When the process is advanced the pleura seldom escapes. The formation of tubercle in the pleura is not common: but inflammation of the membrane is the result of the extension of the pulmonary change, whether of consolidation or excavation, to the surface. The *pleurisy* is often chronic or subacute; if acute, the area invaded at one time is but small. The final result is the

formation of a thick layer of membrane over the affected portion of lung, commonly uniting the organ firmly to the wall of the chest.

This adhesion of the lung has an important protective influence, for, if the process of excavation advances to the surface at a point which is not adherent, the vomica may ulcerate through, and discharge its contents into the pleural cavity, leading, on the one hand, to an acute pleurisy, generally of the purulent variety—*empyema,* or pyothorax; and, on the other, to the entrance of air into the pleural sac—*pneumothorax.*

Another important result of the destruction of tissue is *hæmorrhage:* in earlier stages this follows from congestion alone: in later stages the vessel walls are directly invaded by tubercle, and hence may be eroded; or the vessel wall, weakened by tubercle, dilates so as to form an aneurysm, which may reach the size of a pea or bean, and ultimately gives way at its thinnest part.

Situation of the Lesions.—As already indicated, the above changes follow one another with very varying rapidity, and the spread of the disease through the lung is equally irregular as to absolute time. But the situation of the lesions, and the order of their invasion, are subject to some very constant rules.

The first deposit of tubercle takes place at the apex of the upper lobe; and fresh deposits occur at intervals of weeks, months, or years, lower and lower down. This invasion of fresh parts of the lung takes place by direct contiguity, by lymphatic channels, and largely through the bronchi; infective particles are inhaled into them, and thus start fresh centres of disease. By the time that tubercle forms at the lower levels, the first lesion may have reached the second stage, or stage of consolidation; and later on, when tubercle is being deposited towards the base, the middle part of the lung will have attained the second stage, and the apex the stage of excavation.

Thus one lung may, and frequently does, present all three stages —cavities at the apex; below this, consolidation, with fibrous tissue, pneumonic patches, and caseous tubercle; below this, mostly scattered gray tubercles, with perhaps some congested lung tissue; and below this, finally, some quite healthy lung.

Again, the progress of the disease, while unequal in any one lung, is unequal in the organs on the two sides. As a rule, before a patient dies of phthisis, both lungs are affected, but rarely to the same extent; a large area is commonly involved on one side, before the other is attacked; and so in an advanced case it is common to find the most extensive disease at one apex, and the most healthy tissue, or the only healthy tissue, at the opposite base. The law of the extension of lesions from apex to base may be supplemented by the rule that the apex of the lower lobe is often invaded soon after the apex of the upper lobe, and before the lower part of the upper lobe; and in testing the truth of this observation clinically, it must be remembered that the lower lobe occupies the greater

part of the back of the chest, reaching as high as the third dorsal spine, or the spine of the scapula, and that the greater part of the front of the chest corresponds to the upper lobe. Kingston Fowler has further defined the points at which invasion first occurs as follows: In the upper lobe the disease begins at a point an inch to an inch and a half below the top of the lung, and rather nearer to the posterior and external borders; from this point the disease often extends downwards, by fresh scattered deposits along the anterior aspect of the upper lobe, about three-quarters of an inch from its margin. A less common situation for the first deposit corresponds on the chest wall with the first and second interspaces below the outer third of the clavicle; and from this point the lesions extend downwards and backwards. In the lower lobe the usual seat of invasion is about an inch to an inch and a half below the upper and posterior extremity, and about the same distance from its posterior border; and extension takes place backwards towards the posterior border of the lung, and laterally along the line of the interlobar septum, which position corresponds on the chest to the vertebral border of the scapula when the hand of that side is hooked over the opposite shoulder. A primary lesion of the lower lobe (primary basal phthisis) is very rare.

Changes in Other Organs.—Phthisis of long duration is commonly associated with lesions of other organs, some of which are due to tubercular deposits, while others are of a degenerative kind, and probably the result of the circulation of tubercular or septic toxins. The most constant are tubercular disease of the larynx; tubercular disease of the intestines; fatty infiltration of the liver; and lardaceous disease of the liver, spleen, kidneys, and intestine.

For descriptions of these the reader is referred to the sections dealing with the organs concerned.

At any time, also, in the course of phthisis infection of other organs with tubercle may take place, forming an acute general, or miliary, tuberculosis. In this case the healthy remainder of the lungs, the liver, spleen, kidneys, the cerebral and spinal meninges, and perhaps other parts, are invaded with gray tubercles; and death soon follows, either from tubercular meningitis, or, if the meninges are spared, from impairment of the respiratory function, and from general toxæmia (*see* p. 156). Most of the other tubercular lesions in the body, and many suppurative lesions, are from time to time associated with phthisis, sometimes preceding, sometimes following, the deposit in the lungs; such as caseous or suppurating cervical glands, tubercular disease of the bones, tubercular pyelitis, tubercular peritonitis, scrofuloderma, anal fistulæ, subcutaneous abscesses, &c.

Clinical History.—Pulmonary tuberculosis may run a rapid or a slow course. The most familiar is the *chronic tuberculosis*, which lasts from six months to a few years. The description which follows will mainly apply to this.

The symptoms which characterise phthisis are the following:

Cough, dyspnœa, purulent expectoration, emaciation, hectic fever, and often hæmoptysis.

The commencement is variable. Many cases begin with cough and expectoration of muco-pus or pus, for which no cause can be given, or which is referred to some chill or exposure. Other cases begin with hæmoptysis or spitting of blood. The patient may have been apparently in good health, when, sometimes after an effort, but quite as often when still, or walking or doing something which involves no strain, a tickling is felt in the throat, the patient coughs, and is surprised and alarmed to find that what he spits is blood. Thereupon he may expectorate a few drachms or an ounce, or even half a pint. This may remain as the only symptom, and an examination of the chest may reveal nothing. But after a time, with or without a fresh loss of blood, cough and expectoration supervene, and the case develops like others. In a small number of cases the first apparent departure from health is an acute pneumonic process in one upper lobe, which only partially clears up, while cough and expectoration persist, and the case takes on all the features of phthisis; and in others the first recognisable illness is a pleurisy with effusion, which may even appear to recover completely, and yet be followed by the usual pulmonary changes. Lastly, in some cases indigestion, with loss of appetite, frequent vomiting, and emaciation, are prominent symptoms for some time before the special indications of a lesion of the chest are apparent.

The disease is also very variable in its course in different cases. Patients with the earliest symptoms, whether hæmoptysis, or cough or wasting, placed under favourable conditions of climate and hygiene, may completely regain their health; and it has long been known that in persons killed by accident, or dying of disease unconnected with the lung, cicatricial and pigmented patches, with perhaps calcareous deposits, are found in the apices, which can only be regarded as the remains of former tubercles.

If, however, it is well established before being submitted to treatment, the result cannot be so satisfactory. Thus the disease may be fatal in three or four months, or it may last twelve or fifteen years before finally killing the patient; and in this time its progress will be very unequal, often quiescent for months or a year or two, and then making great strides, with hæmoptysis or much fever. While the more rapid cases are fatal chiefly by the extent of lung involved, the cases of longer duration threaten life by a number of complications, some of which are lesions of the lung itself, such as hæmoptysis, pneumothorax, empyema, and bronchitis; while others involve distant organs, such as tubercular meningitis, ulceration of the intestines and diarrhœa, nephritis, and lardaceous disease of the viscera.

Local Symptoms.—These will now be described somewhat more in detail.

Cough.—This is a very common symptom, and generally, though not always, present as long as the disease is in any degree active.

It is mostly easy at first, sometimes not much more than a clearing of the throat; it becomes harder and more painful in the later stages; and with extensive cavities it occurs in prolonged attacks, painful to the patient, distressing to those about him, and lasting perhaps more than a minute, until at length some sputum is brought up. With laryngeal complication the cough acquires a hoarse or husky quality.

Dyspnœa.—Shortness of breath is often early noticed, and becomes very marked as more and more of the lung is diseased, and so the surface available for aeration of the blood is diminished.

Expectoration.—In the early stages this is not different from the sputum of bronchitis—that is, it is either simply mucous, or it is muco-purulent; and this is accounted for by the bronchitic processes that frequently accompany phthisis. But sometimes comparatively early, and always in later stages, the sputum becomes purulent, of green or greenish-yellow colour, opaque, and quite free from air-bubbles. If it is very fluid the individual sputa may run together and lose their separate form; but the sputa of phthisis often keep separate long after expectoration, and, from the round, flat shape that they assume in the sputa-vessel, they are called *nummular*, or coin-shaped. This is no doubt due to the accumulation of the secretion in cavities in the lungs, and hence it constantly occurs in phthisis, but may also be present in cases where the cavities are produced by dilated bronchi (bronchiectasis).

Phthisical sputa, examined under the microscope by suitable methods, are found to contain pus, mucus, and blood-corpuscles, drops of myelin, pavement epithelium from the mouth, alveolar epithelium from the lungs, tubercle-bacilli, and in the destructive stage elastic tissue from the walls of the air-vesicles (*see* p. 534).

Hæmoptysis.—When hæmoptysis occurs as the first sign of pleurisy, the blood is generally bright red and frothy; it is expectorated in variable quantities, and, as a rule, for some hours or days the patient continues to spit pellets of blood which have a darker and darker colour, become gradually less frequent, and then cease entirely. There may at this time be no other sputum. In later stages, when the disease is well established, the muco-purulent or purulent sputum is often streaked or stained with blood. A few streaks in the sputum may proceed from small vessels in the bronchial mucous membrane, but more characteristic of phthisis is the intimate mixture of bright blood with the sputum, or the discharge of pellets of coagulated blood frequently during the day. From time to time may occur more abundant hæmorrhages, like those first described, in which the blood comes up apart from the ordinary secretion; and if a large vessel is ulcerated, or what is more often the case, if a small aneurysm in a cavity ruptures, several ounces or a pint or two of blood may be discharged within a short time, and death may follow rapidly.

Physical Signs.—These are best considered in reference to the three different stages of phthisis; but all these stages may be present

at the same time in the same lung, and the process is most advanced at one or other apex.

In the *first* stage (tubercular deposit) the physical signs may be very slight, and vary considerably in different cases. The eye or the hand may detect a slight impairment of mobility on the affected side. For this purpose one hand should be laid on either chest just under the clavicle, and the relative movements should be watched during tranquil and during full, yet gentle respiration. Careful percussion of the apex may give only slight impairment of the note as compared with the opposite side; this may be found just below the clavicle, or on the clavicle, or in the supra-clavicular fossa, or it may be behind, above the scapula. Below the clavicle there may be tenderness on percussion. Auscultation often gives much more certain indications, especially a diminution of the vesicular murmur, and the presence of fine or medium râles during inspiration. Sometimes there is nothing but the deficiency of vesicular murmur, but this, if associated with impaired resonance or mobility, is of much importance. The inspiratory murmur may be irregular, jerky, or wavy—the so-called cog-wheel respiration; or it may be roughened; or the expiratory murmur may be loud and prolonged, assimilating the respiration to bronchial breathing, and this may be associated with an increase of vocal resonance. It is, however, very important to remember that prolonged loud expiratory murmur with loud vocal resonance is not uncommon upon the right side in healthy individuals, and especially in females. And, as a rule, repeated examinations at short intervals are needed before one can with confidence state that there is evidence of phthisis from the physical signs, although cough, expectoration, wasting, and febrile reaction may justify the strongest suspicions. Râles with deficient vesicular murmur are the most trustworthy signs.

In the *second* stage (consolidation) the physical signs are in many respects similar to those of the second stage of pneumonia. According to the extent of lung involved, there is more or less impairment of mobility of the affected side; and when the progress has not been unusually rapid, there is obvious depression of the supra-clavicular and infra-clavicular regions, caused by contraction of fibrous tissue, or perhaps by the earliest destruction of tissue, producing cavities as yet too small to be recognised by physical signs. On percussion, there is increasing loss of resonance as the case goes on; but the dulness is rarely so absolute as that which occurs over a pleural effusion; and sometimes it has a high-pitched, boxy, or more tympanitic character. On auscultation, bronchial breathing of different qualities and pitch is heard, and the voice and cough are loudly bronchophonic. Râles are often present; they are mostly of sharp clicking, or consonating character.

It is in the *third* stage (excavation), when the disease has existed some time, has seriously involved one lung, or has already attacked the other, that one can best recognise the modifications in the shape of the chest, which may have already commenced in preceding

stages. The chest takes on the type of extreme expiration. It is flat, long, and narrow; the shoulders are depressed and sloping; the lower ribs come within a short distance of the crest of the ilium; the upper ribs in front are wide apart, the lower ribs are crowded together, and the costal or epigastric angle is reduced to its smallest size. The nipple tends to lie high in relation to the ribs—for instance, in the third space—while the heart may strike the fifth rib, instead of the fifth space; as if the ribs had glided down between the skin and the viscera. In addition to this general change in the chest, there is retraction of the upper part of the chest on the most affected side, and a corresponding impairment of movement. At this point, which we now suppose to be the seat of cavities, we find that the percussion note is variable. It must be remembered that the cavities form in lung which has first become solid. Now, absolutely solid lung gives dulness on percussion, and a lung entirely hollowed out into one large cavity gives a resonant note; the percussion note over excavated lung must, therefore, vary with the size of the cavity, its nearness to the part of the chest percussed, the amount of consolidation around it, or between it and the point struck, and the degree to which the ribs are fixed by pleuritic adhesions. The note may, therefore, be quite dull, or of tympanitic resonance; more often it is dull to light percussion, and of different degrees of boxy, or high-pitched, half-resonance on a heavier stroke being used. If there is a large cavity in free communication with the bronchial tube, and the patient's mouth be open, percussion will often elicit the *cracked pot sound* or *bruit de pot fêlé*, which is somewhat, but not exactly, like the sound produced by striking the two clasped and hollowed hands upon the knee to delude children with a hope of pence. The resonance over such a cavity is raised in pitch when the patient opens his mouth (Wintrich); and during inspiration, while it falls in expiration (Friedreich); and the pitch varies with change of position of the patient (Gerhardt). On auscultation over cavities, one may obtain hollow bronchial, or cavernous or amphoric breathing, according to the varying degree of excavation and condensation around. It is only truly amphoric when the cavity is very large indeed. The vocal resonance may be simply increased (bronchophony), or it may be pectoriloquous as well, the whispered voice being transmitted with unusual distinctness; or pectoriloquy may be present alone. In extensive cavities, when the patient speaks, there is heard, in addition to the loud vocal resonance, a kind of *whispering echo* of the same, apparently produced by reverberation from the walls of the cavity. Bubbling râles of large size, and the peculiar phenomenon known as metallic tinkling, are often heard in cavities. The auscultatory sounds are often best brought out on deep inspiration or on coughing; when, sometimes, post-tussive suction may be heard. They may all be absent if, and as long as, the bronchial tube in connection with the cavity is blocked. Moreover, a cavity probably does not give distinctive signs as compared

with consolidation, unless it has reached a large size, about that of a walnut.

In cases of long standing, in which the left lung is mainly affected, the contraction of that organ allows the heart to come more fully into contact with the chest-wall, and in the second left intercostal space may be observed the pulsation of the conus arteriosus of the right ventricle; the closure of the pulmonary valves may be then felt with the finger, and the second sound is unusually accentuated, or rather it is heard with greater distinctness than is normal.

General Symptoms.—The more important are pyrexia, with night sweating; loss of flesh and strength; anæmia; and the evidences of imperfect aeration of the blood.

Pyrexia.—From the earliest days of phthisis, fever may be present, but it generally bears some relation to the activity of tubercular and pneumonic processes in the lung, so that if the mischief becomes inactive from time to time, the fever may for a corresponding time be absent; but it is often present continuously for months. The temperature is commonly higher in the evening than in the morning, and is either remittent or intermittent in type. In the former it may be 99° to 100° in the morning, and 102° or 103° in the evening; in the latter it is 98·4° or even lower in the morning, and reaches 100° to 103° in the evening. The lower degrees of fever are often not appreciated by the patient; the higher are accompanied with the discomfort and malaise common in pyrexia, and especially in the advanced stages of the disease, with profuse sweats, and even slight chill before the sweating. Actual rigors, however, are exceptional, and the most common event is for the patient to sleep more or less tranquilly in the early part of the night, so far as the cough will let him, and to wake up in the early morning to find himself drenched with perspiration. These are the colliquative sweatings of older writers. Some night-sweating is not uncommon even in early stages.

Loss of Flesh and Strength.—Emaciation is the rule in phthisis; it may be one of the earliest symptoms, and may give a note of warning, when the cough has been thought to be a mere bronchial catarrh; towards the end of a chronic case the emaciation is extreme. If, as a result of treatment at any time, the patient improves, he commonly puts on a little flesh, or at any rate remains stationary. Exceptionally, nutrition is maintained fairly well, even when the physical signs show that there is a considerable and even apparently active lesion. The muscular power is soon enfeebled, and the patients lose energy, becoming languid and unfit for prolonged exertion, whether of mind or body. The mental condition in many patients, however, is one of great hope and confidence; even when helpless in bed they often fail to realise how ill they are, and look for complete recovery could they but once get rid of the cough.

Anæmia.—The loss of nutrition is represented also in the blood, and the patient is commonly pallid, both in the early and late

stages. But this anæmia is often masked by two other conditions —one is the hectic fever, which leads to a flushing of the cheek and lips, especially in the evening; the other is the imperfect aeration of the blood causing cyanosis.

Imperfect Aeration of the Blood.—This shows itself by cyanosis of the face, especially in acute cases involving a large area of lung, and in chronic cases in which the right side of the heart has become somewhat dilated. Another condition which is usually attributed to a retarded venous circulation consequent upon bad aeration, but of which the mechanism is not quite clear, is that of the *clubbed fingers,* or *ungues adunci.* The finger loses its tapering form, and the last joint becomes thickened, especially from palmar to dorsal surfaces, but also transversely. The nail is unusually convex from base to tip, and seems to curve over the end of the finger. The appearance is more pronounced on account of the wasting of the rest of the finger. It is a very common feature of phthisis, not peculiar to it, but present in a large proportion of the cases. The same change may be seen in the toes.

Complications.—It will not be necessary to do much more than enumerate the various complications that occur in phthisis, as they are described in different parts of this volume under the particular organs concerned. Many of them result from the formation of tubercles in other parts of the body. They are more common in the long-standing chronic cases.

Respiratory Organs.—Tubercular disease of the larynx (*see* p. 466) is common in phthisis, and considerably aggravates the patient's distress. In exceptional cases the symptoms of laryngeal mischief are obvious before those of the pulmonary lesions, but it is doubtful if the tubercle is ever actually deposited in the larynx before it is formed in the lung. Very rarely indeed does this complication either directly cause death or require tracheotomy.

Pleurisy.—This is so common as to be almost a part of phthisis. An old phthisical lung is usually adherent to the chest by a thick fibrous layer, and pleuritic lymph forms nearly always over tubercular lesions that approach the surface. Sometimes liquid is poured out, and this is mostly a sero-fibrinous effusion, less often an empyema.

Pneumothorax.—This occurs in a very small proportion of cases, but phthisis is by far the commonest cause of pneumothorax. If the liquid contents of a cavity escape into the pleura, an acute pleurisy may be set up, effusion takes place, and there will not only be a pneumothorax, but a *pyo-pneumothorax,* or less often a *hydro-pneumothorax.* Pneumothorax in these different forms may be quickly fatal, or it may slowly disappear, or even while persisting may not very seriously increase the respiratory difficulties of the patient (*see* p. 557). *Hæmothorax* is a rare complication.

Circulatory System.—The heart wastes in chronic phthisis, but not to the same extent as in a fatal case of cancer. In some more chronic and fibroid forms the right ventricle is dilated and

thickened to a moderate extent. The arterial erosions and small aneurysms of the pulmonary artery in cavities in the lungs have been already mentioned.

Femoral thrombosis is frequent in the last days of patients suffering from phthisis; it is more common on the left side.

Alimentary System.—*Parasitic stomatitis,* or thrush, occurs in the same circumstances as femoral thrombosis.

Anorexia, indigestion, nausea, and *vomiting* are common accompaniments of phthisis more or less during its whole course. A capricious appetite and a distaste for fat in every form have been noticed even before definite symptoms in the lungs. In the last stages sickness or loathing for food is so marked, that it is one of the chief difficulties of doctor and nurse to get the patient to take anything at all. The most extraordinary things are fancied by the patient at one moment, only to be rejected directly they are put before him.

Diarrhœa is common in late stages; it may be due simply to catarrhal conditions, to tubercular ulceration of the ileum, or to lardaceous disease. The stools are variable; sometimes yellow in colour, and containing a little mucus, or blood. Large hæmorrhages are not often seen.

Peritonitis is very rarely the result of perforation of a tubercular ulcer. More often it is due to tubercles in the peritoneum, but it is not a common complication.

Lardaceous disease of the liver, spleen, kidneys, and intestines occurs in numerous cases of phthisis—about 20 per cent. (Powell).

Fatty liver is somewhat more frequent.

Tubercular disease of the *epididymis* and *vesiculæ seminales* in men, and of the *uterus* and its *appendages* in women, occasionally occurs, but these lesions do not usually form prominent complications at the bedside. *Fistula in ano* is occasionally associated with phthisis.

Nervous System.—Here also tubercle occurs, generally as tubercular meningitis, which is a cause of death in a small number of cases. This secondary tubercular meningitis is often very rapid; perhaps the earlier indications are lost in the general symptoms already present.

Pains are frequent; those about the chest are attributed to pleurisy; others occur in the limbs, and *peripheral neuritis* may affect the nerves of the extremities.

Addison's disease of the suprarenal capsules is sometimes associated with phthisis.

Acute nephritis, apart from tubercles or lardaceous disease, is an occasional complication.

General tuberculosis is one of the fatal complications of phthisis. The lungs, liver, spleen, and kidneys are the organs commonly affected, and sometimes the cerebral and spinal meninges (*see* p. 154).

Bones and Joints.—Tubercular disease of joints, caries of ribs

with subcutaneous abscesses, caries of the spine with psoas abscess, and other similar lesions may coexist with the pulmonary disease.

Fatal Termination.—Death takes place in the following ways: Exhaustion, hæmoptysis, pneumothorax, meningitis, perforative peritonitis, and uræmia.

Exhaustion, or, to speak in more modern terms, the disturbance of all the functions and finally of the circulation, from poisoning by the toxins of tubercular and pyogenic organisms, accounts for the larger number of cases. The collapse is explained by the pyrexia, the loss by expectoration, sweating, and diarrhœa, and the deficient supply of nutriment from anorexia, nausea, and vomiting. Occasionally there is sudden collapse in a patient actually going about, and this may be mistaken clinically for the result of pneumothorax or of hæmorrhage.

Varieties of Phthisis.—The forms commonly described are pneumonic, catarrhal, tubercular pneumonic, tubercular, fibroid, hæmorrhagic, and laryngeal. Of these, pneumonic, the most acute, and fibroid, the most chronic, are the varieties which stand out most distinctly and require separate notice here.

Pneumonic Phthisis (*Scrofulous Pneumonia*).—This begins very much like an attack of acute pneumonia, with pain in one side, high fever, chills, and night-sweats, cough and expectoration. The physical signs also are those of pneumonia; but they are most marked at the apex, and spread downwards. Dulness, bronchial breathing, and bronchophony are accompanied by coarse mucous râles, consonating râles, and loud clicks. Often the condition is much more marked in one lung than in the other. The mischief extends rapidly: the pyrexia is severe, there are profuse sweats, appetite is entirely lost, and prostration becomes extreme. The indications that the lung is breaking down are more and more marked; the temperature assumes an intermittent type, the sputum is purulent, and contains *débris* of lung-tissue. The illness is often fatal in the course of from five to twelve weeks, either by exhaustion, or by hæmoptysis, which is always abundant if it occurs at all; or by pneumothorax from the opening of one of the rapidly formed cavities into the non-adherent pleura. The lungs are solid from combined hepatisation and caseation; and there are numerous small cavities in every part of the organ, with ragged irregular walls, and purulent contents. In this pneumonic and caseous material definite miliary tubercles cannot be found; but bacilli have been often seen in such lungs.

Occasionally the process may not be at once fatal; partial recovery takes place, and the patient lingers on for some years.

Fibroid Phthisis.—This form may supervene upon chronic pleurisy and chronic pneumonia, and occurs in association with the prolonged irritation of the lungs among workers in certain trades. It is extremely chronic, and often affects one lung only. Clinically, the case is distinguished by the evidences of contraction of the diseased lung; the chest is sunken, the heart is displaced to the affected side, the opposite

lung may extend its resonant area in the same direction; the spleen and stomach if the left lung is diseased, or the liver if the right, may be drawn far up into the chest. The physical signs of cavities are chiefly at the apex, as in other cases of phthisis; but impaired resonance, bronchial breathing, and bronchophony are perhaps present over the whole of the affected lung. If the other side is involved, it is only at the apex. The symptoms are cough, purulent expectoration, and dyspnœa; the cough is frequently hard and prolonged, and the sputum may be fœtid from retention. There is, as a rule, no constitutional disturbance; there is no sweating, and the temperature is normal. After some time the right ventricle of the heart becomes dilated, and cyanosis and dropsy ensue. Lardaceous disease may result from the continued profuse discharge, and diarrhœa and albuminuria assist the fatal termination. Hæmoptysis occurs, but is not constant. *Post mortem*, the lung is found to be contracted to one-third or one-quarter its normal size, firmly adherent to the chest by a thick dense fibrous layer, and presenting a quantity of dense white or gray fibrous tissue, which contains caseous or cretaceous deposits, vomicæ, and dilated bronchial tubes. Tubercular or fibroid change may be present to a smaller extent in the opposite lung.

Diagnosis of Phthisis.—In advanced cases the physical signs of consolidation or cavity at the apex of one lung, with purulent or sanguineous sputum, fever, and emaciation, determine the diagnosis, especially if the fever is intermittent or decidedly remittent, with night-sweats, and if the illness is of some duration—that is, longer than a pneumonia. Except in the earliest stages, and in periods of quiescence or arrest, the sputum will show *tubercle-bacilli*. For their detection they require to be stained, and to be submitted to a microscopic power of 350 or 400 diameters. The Ziehl-Neelsen method of staining is now commonly employed. A cover-glass is smeared over with a thin layer of sputum, and is passed three times through the flame of a spirit lamp to coagulate the albumen. A solution of 1 part of fuchsin in 10 parts absolute alcohol is added to 100 parts of a 5 per cent. aqueous solution of phenol. This is heated till the steam rises; and the cover-glass is floated on it, film downwards, for three or four minutes, rinsed in water, and immersed in a 20 per cent. solution of sulphuric acid until it is decolourised. It is then washed in water, and counterstained by means of a nearly saturated watery solution of methylene blue, again washed quickly in water, dried, and mounted in xylol balsam.

The fragments of *elastic tissue*, which are present with sputum in the later stages, may be seen with the microscope either by teasing out the little tough nodules which are sometimes found, or by boiling the sputum in liquor sodæ for twenty minutes, and examining the sediment. Elastic tissue is also found, however, in the sputum of pulmonary gangrene.

It is much more difficult to be certain of the presence of phthisis

PLATE I

Skiagram of a case of phthisis, showing dark mottling of both upper lobes. A lighter area above the right clavicle is due to a cavity. (Taken by Dr. A. C. Jordan.)

[*To face p.* 535.

in its early stages. Cough, expectoration, wasting, evening rises of temperature, and even hæmoptysis often precede the physical signs by weeks or months; and here again conclusive evidence may be afforded by the detection of bacilli in the sputum, either by the staining method above described, or, failing that, by inoculation of a rabbit.

The first *physical signs* of any value in diagnosis are impaired resonance at one apex with diminished vesicular murmur, or diminished vesicular murmur with râles on inspiration or on coughing. The *Röntgen rays* may give valuable help by the discovery in early stages of a dark area at one apex, with limited movement of the diaphragm on deep inspiration (*see* Plates I. and II.).

The measurement of the *opsonic* quality of the patient's blood-serum in relation to tubercle-bacilli is also used as a means of diagnosis. The opsonic index is taken in the manner previously described (*see* p. 20), and it is found that in cases of phthisis this index is rarely normal: it is either decidedly below unity (·6 or ·7), or much above it (1·3 or 1·4). In the former it is assumed that the resisting power of the body to tubercle is slight; in the latter that the resisting power is in excess of the normal because it has been stimulated by excess of toxins caused by active disease.

Koch's *tuberculin* may be employed for purposes of diagnosis in four ways: (1) Subcutaneously, in order to produce *reaction*; (2) subcutaneously, to affect the opsonic index; (3) by application to the conjunctiva (Calmette's *ophthalmic reaction*); and (4) by application to the skin (Von Pirquet's *cutaneous reaction*).

(1) When tuberculin is injected in certain doses into tuberculous individuals, it causes a rise of temperature, which does not occur in the healthy. For diagnosis it must be used only in cases in which there is no fever, no advanced disease, and no secondary infection. A rise of temperature of one degree within thirty-six hours is all that is sufficient; and no greater effect than this should be sought. The tuberculin is diluted with a ·5 per cent. solution of carbolic acid; and in a delicate subject an amount equal to one milligramme of tuberculin (Latham) or less is injected into the back or buttock. If in three days there has been no rise of temperature a double dose (2 milligrammes) is injected; if there is no reaction a larger dose (3 milligrammes) is injected after another interval of three days; and then, failing any reaction, tuberculosis is excluded. For more robust patients the successive doses may be 1, 5, and 10 milligrammes, or 5, 10, and 20 milligrammes.

(2) In the healthy subject, the injection of a small dose of *tuberculin* ($\frac{1}{2000}$ mg. to $\frac{1}{5000}$ mg.) lowers the opsonic index (*negative phase*) for a day or two; and it then rises slightly above the normal (*positive phase*), and then returns to normal. In a tubercular subject the negative phase is of much longer duration, seven days or more, and it takes two or three weeks for its return to its usual standard.

(3) A few drops of a ·5 per cent. solution of tuberculin are allowed to fall on the conjunctiva near the inner angle of the eye, and the

eyelids are kept apart for a few moments. If the subject is tuberculous in any part of the body whatever, the conjunctiva of the lower lid and the caruncula will begin to redden in three hours, the injection increases in six hours, the caruncula is swollen, tears flow freely, and the eye is covered with slight exudation. The reaction reaches its maximum between the sixth and the thirteenth hour, and subsides entirely in two, three, or more days.

(4) The test for the cutaneous reaction consists of one part of Koch's old tuberculin (O.T.) with one part of a 5 per cent. solution of carbolic acid in glycerin, and two parts of normal saline solution. A control is a similar solution of carbolic acid in glycerin or saline. The skin is "vaccinated" in two places in the same way as in vaccination for small-pox (*see* p. 78); in one spot with the tuberculin solution, in the other with the control. Reaction is shown by hyperæmia with some œdema within twenty-four hours; in forty-eight hours the spot is reddened, slightly raised, and palpable; on the third day the spot begins to fade, and on the fourth day it is gone.

Before *hæmoptysis* is regarded as indicative of tubercle, it must be clearly made out that the blood really comes from the lung, and not from the stomach. The descriptions of a patient are often very unsatisfactory or misleading. Blood from the lungs should be coughed up, and should be bright red and frothy; it is often preceded by a tickling at the throat, not by a sense of nausea. *Anæmic* girls often speak of finding blood on their pillows on waking. This is, no doubt, from the gums or cheek, is generally diluted with saliva, and may be mixed with buccal epithelium. *Malingerers* may produce blood in a similar way. In *purpura*, blood may be expectorated actually from the lung, but the cause will be readily distinguished by the associated symptoms. Hæmoptysis is frequent in *heart disease;* especially early mitral stenosis in young people may give rise to cough, hæmoptysis, and shortness of breath, and the heart should therefore always be examined.

Phthisis is sometimes masked by *bronchitis*. The accentuation of the physical signs at one or other apex should help one, as well as the history of the illness, hæmoptysis if present, and the detection of bacilli in the sputum. The possible confusion with *bronchiectasis* has been mentioned (*see* p. 493). Phthisis may be sometimes simulated by *empyema*, which will produce fever, sweating, emaciation, cough, and purulent sputa; but examination will show the predominance of physical signs at the base in empyema, while the apex is clear. The two may, of course, co-exist, and in any case exploration with a needle and syringe will settle the point. *Pleuritic effusion* sometimes causes a tympanitic note under the clavicle, with bronchial breathing and bronchophony, and these with dyspnœa and fever may raise a suspicion of phthisis. The history and the physical signs of effusion in the lower part of the chest should make the case clear.

Prognosis.—In scarcely any disease are cases so different as they may be in phthisis. Discovered in its earliest stage it may be

Skiagram of a case of phthisis, showing a dark area in the right upper lobe due to consolidation, and a light area above the clavicle due to a cavity. The left lung is healthy. (Taken by Dr. A. C. Jordan.)

[*To face p.* 536.

cured so completely that no trace of it can be detected clinically; it may in other cases be fatal in a few months; or it may last ten, twenty, or even fifty years with obvious physical signs and symptoms throughout. The variable elements are the virulence of the infection, and the capacity for resistance on the part of the patient; and it is difficult to estimate which will gain the upper hand until the patient has been under observation or treatment for some time. Improvement may occur at once under treatment; or, at any time in the course of the illness, the protective powers of the patient may be so increased as to cause arrest of the process for a long period. And in no case should hasty predictions be made as to when the end will take place. Alcoholism, the occurrence of other infections, overwork, anxiety, and defective hygienic conditions are certainly unfavourable and likely to induce a rapid course of the tubercular disease. Much fever, free hæmoptysis, abundant expectoration, and physical signs indicating rapid changes in the lung, which can best be estimated from two or more examinations at a few weeks' interval, are also unfavourable.

Treatment.—Attempts have been made to cure phthisis by agents which will destroy the bacillus. Such an attempt was the injection of Koch's *old tuberculin* in 1890–91, for it was thought that the increased local changes which accompanied the febrile reaction would damage the bacilli; but the method proved to be useless and dangerous. Since then creosote, guaiacol, and guaiacol carbonate have been largely used, and are believed to have done much good in some cases. They may be given in increasing doses up to large amounts. From 10 to 100 minims of creosote in capsules or dissolved in cod-liver oil, 10 to 60 minims of guaiacol in capsule, or 20 to 90 grains of guaiacol carbonate in cachet or wafer can be given daily. Antiseptic drugs have also been administered by more or less continuous inhalation from a Coghill's or Burney Yeo's respirator worn over the mouth or nose. Dr. Lees advises the continuous use of it day and night, waking or sleeping, except when at meals. His solution consists of carbolic acid 2 parts, creosote 2, tincture of iodine 1, spirit. ætheris 1, and spirit. chloroformi 2 parts. Of this, 6 or 8 drops are poured on the inhaler every hour during the day, and two or three times during the night.

But our chief reliance must be on the improvement of the body and its tissues in every possible way, so that it may be enabled to resist the inroads of the disease, or rather that the tissues may become less fitted as a soil for the bacillus, and less readily excited to inflammation in its different forms. This indication is met by providing for the individual perfect hygienic surroundings, pure air, and sufficient good food.

Treatment with the above objects should be taken *as soon as possible* after the evidence of tubercular infection is confirmed. In this *early stage* three methods are available, namely: Change of climate, a sea voyage, or residence in a sanatorium.

Change of Climate.—The places usually selected are South Africa,

New Zealand, high altitudes in Switzerland such as Davos, Wiesen, and Maloja, or the English South Coast, at Hastings, Dover, or Folkestone. He may in one or other of these places obtain a pure and bracing air, which he can enjoy for several hours daily outside the house, without the risk of catching cold, and without undue strain on the lungs; and he may spend the whole of the winter avoiding the cold, damp, and fog of that season in the greater part of England, and returning to his home in the summer when the weather is more tolerable. With the advent of winter he must again seek the climate which he has found suitable.

Sea Voyage.—A sea voyage of three or six months' duration on a sailing-vessel has often been of the greatest benefit, providing a pure air, and allowing the freest exposure of the patient without risk. But there are disadvantages about steam vessels; and in any vessel the confinement to a small cabin at night is undesirable, there may be difficulties in the supply and cooking of food, and with prolonged bad weather the conditions may be the reverse of satisfactory. It is only cases in the very earliest stage, and without hæmorrhage, that can be safely sent on long voyages.

Sanatorium Treatment.—This is an endeavour to carry out the indications above mentioned in the patient's own country; and it meets the requirements of the thousands who, on the score of expense or for other reasons, cannot possibly travel or reside in distant countries. The object in view is to provide that the patients, though kept warm and sheltered from rain and cold winds, shall live night and day practically in the open air. The day is spent as much as possible outside the building, either in the open, or in shelters screening the patient from the wind, and when possible exposed to the sun. The bedrooms and day rooms are thoroughly ventilated, with the avoidance of draughts, so that the air within is as pure as that outside, and the rooms are constructed so as to prevent any accumulation of dust. The patients are well fed, having three meals daily of plain but varied food. Exercise is only permissible when the morning temperature is normal, and the evening temperature not above 99·5°: the exercise will probably raise the temperature for a time, and if it is raised above 100·5°, the exercise is stopped. The patient walks slowly and is not allowed to talk. In any case, he rests one hour before meals, and one hour after: and violent exercise and indulgence in exciting games or recreations are prohibited. The clothing of the patient must be suited to the temperature of the air. Treatment on this system has benefited many patients temporarily: but it must be submitted to for very much longer than the period of three months for which it is sometimes prescribed. Even where it cannot be continued, it is useful for the patient to have undergone it, in order that he may at his own home carry out the principles as far as circumstances will permit.

Further help in increasing the patient's resisting power is sought by the use of *tuberculin* in combination with observations on the opsonic index. If the index is found to be low, an injection of

$\frac{1}{1000}$ mg. or $\frac{1}{2000}$ mg. is made, and the index is again taken after three or four days. The negative phase is allowed to pass, and when the positive phase is established—that is, after two or three weeks—another injection may be given. The injections may be repeated at intervals, but never at a time when the index is already depressed by a preceding dose. These injections do not cause any rise of temperature, or any *reaction* of the kind produced by the larger doses mentioned under Diagnosis.

This method of treatment is still on its trial as applied to pulmonary tuberculosis.

Graduated labour.—Dr. Paterson has shown at the Frimley Sanatorium that by graduated exercise and manual labour, patients with early, or not very active later, lesions, in whom the temperature is less than 99° F., may be considerably improved not only in general health and strength, but also as regards their local signs. Each case must be treated on its merits, and the work ranges from a half-mile walk daily, to six hours' spade-work or load-carrying in the day. Beginning with the lightest exercise, the patient is gradually put to heavier and heavier work. A rise of temperature to 99° F., and headache are the indications that the work is too much. Dr. Inman shows from examinations of the opsonic index, that the exercise and work cause an auto-inoculation with tuberculin, which as long as it is kept within limits, is probably the cause of the improvement.

In connection with sanatorium treatment may be mentioned the "class system," devised by Dr. Pratt, of Boston, U.S.A., which is a method of organising in the homes of the working classes, for patients who cannot go into hospital or have been discharged relieved, a system of free ventilation by open windows, together with an abundant diet of milk and eggs.

In *advanced stages* the above methods of treatment are much less applicable; and the patients must be treated as invalids. In markedly hæmorrhagic cases, and in cases complicated by rheumatic tendency, feeble circulation, bronchitis, emphysema, or albuminuria, the bracing climates and high altitudes are likely to be injurious; and patients in the third stage are more often benefited by a warm and rather moist climate, combined with pure air, such as is to be found at Bournemouth, Torquay, Penzance, and neighbouring places in England, and abroad at Mentone, Cannes, San Remo, and the Riviera generally. Other places south of England do good service in this way, such as Algiers, the Canaries, and Egypt. But in all cases the principle of pure air, by means of free ventilation, should be as far as possible carried out.

Under any circumstances the diet should be plain, nutritious, and abundant; stimulants are rarely necessary. Nutrition may be furthered by cod-liver oil, taken in doses of 2 to 4 drachms two or three times a day after meals, and by quinine and iron in small doses so long as the digestion is perfect. Defective action on the part of the stomach must be at once met by suitable remedies (*e.g.*, alkalies and nux vomica), and the importance of good gastric

digestion as an aid to nutrition must never be lost sight of in the treatment of the various symptoms and complications.

No very satisfactory results have been derived from the *surgical* treatment of phthisical cavities.

Symptomatic Treatment.—During treatment in a sanatorium, drugs are avoided as much as possible; and it is generally found that the symptoms disappear with the improvement of the patient. It is in all cases essential that the digestion should not be upset by the medicines administered.

Cough.—The rapid diminution of cough has been constantly observed in the open-air treatment. Only if it is painful, frequent, or keeps the patient from sleep, may it be treated by small doses of opium or morphia, in combination with expectorants; for instance, tr. camph. co. with tr. scillæ, or liq. morphinæ hydrochlor. with vin. ipec., or syr. papav. with camphor water, or a few minims of chlorodyne. Counter-irritation by tincture of iodine or by small blisters is also useful. A morning cough, which gets rid of accumulated secretion, may be usefully promoted by a little ammonium carbonate.

Night Sweating.—This can be generally checked by 1 minim of liq. atropinæ sulph. given in a little water at night; or 2 or 3 grains of oxide of zinc in a pill, with or without $\frac{1}{6}$ grain of extract of belladonna. Arseniate of iron ($\frac{1}{5}$ grain) or picrotoxine ($\frac{1}{50}$ grain), or tincture of nux vomica may also be used.

Hæmoptysis.—The patient should be kept in bed in the semi-recumbent posture, an ice-bag should be placed on the front of the chest over the lung from which the blood is believed to come, the diet should be fluid, cold, and given in small quantities at a time, and one of the following styptics should be ordered: Opium with dilute sulphuric acid; pil. plumbi cum opio; digitalis, opium, and quinine—a grain of each; calcium chloride in 5 or 10 grain doses; or tr. hamamelidis. Bleeding often ceases after inhalation of a few drops of amyl nitrite. A small injection of morphia at first will serve to quiet the patient, who is frequently excited and alarmed.

Diarrhœa.—For this, one must carefully regulate the diet, and use the vegetable astringents, mineral acids, opium, sulphate of copper in $\frac{1}{4}$-grain doses or subnitrate of bismuth. Methylene blue has been recommended in doses of 1 grain three times a day. The patient should be told that the urine will be turned blue or green.

Laryngeal Ulceration.—(*See* Tubercle of the Larynx, p. 466.)

Pleuritic pains are frequent, and are often relieved by painting the surface with tincture of iodine. Anodynes internally may be necessary. Many believe that pleuritic effusion delays the progress of the disease in the corresponding lung, and postpone tapping until pressure is extreme. An empyema requiring evacuation may be aspirated.

Pneumothorax.—For the acute pain, on its first occurrence, a morphia injection may be required. Later on, the complication may cause but little trouble, and may be left alone. If air accu-

mulates so as to give serious trouble from distension, it should be let out by a trocar and cannula.

Excessive Expectoration.—This condition is undoubtedly benefited by the method of antiseptic inhalation which has been described in connection with direct treatment (*see* p. 537). Eucalyptol and thymol may be used with one or other of the drugs there mentioned. The patient wears the respirator for one, two, or three hours at a time, and breathes the vapour into his lungs. As a consequence, the expectoration often becomes less abundant, and loses its offensive characters; the patients have gained weight, and improved in their general health. Mendel's tracheal injections (*see* p. 494) may be employed for the same purpose.

Prevention.—The rules of life which are recommended for a patient with early phthisis apply to children born of phthisical parents, at least as long as they can be enforced; it is by such means that they can best unfit their tissues for the reception of the bacillus. Phthisical persons about to marry should be informed of the risk that their offspring may develop the disease; a risk which is thought to extend to the marriages of healthy persons, if both come of a phthisical stock. One must not forget the possible conveyance of the disease from person to person in some circumstances, especially its indirect transmission in the manner suggested (*see* p. 152) between relatives sleeping together, or brought into constant close contact, as when one dying of phthisis is assiduously nursed by another. The phthisical should not sleep in the same room with a healthy person. Underclothes and bed-linen should be scalded before being washed. The sputa should in all cases be ejected into antiseptic fluid (5 per cent. carbolic acid solution); and they should finally be rendered innocuous by exposure to boiling water for ten minutes. Tuberculous mothers should not suckle their infants.

PULMONARY ASPERGILLOSIS

The lungs are affected with local caseating or suppurative lesions as the result of glanders (*see* p. 185), of actinomycosis (*see* p. 191), and of a fungus, the *Aspergillus fumigatus*, of the order Ascomycetes. This rare disease occurs amongst pigeon-feeders, in consequence of the fungus being contained in the seed, which the feeder puts into his mouth for transference to that of the pigeon. The lesions are like those of tubercle, with cavities, fibroid changes, or emphysema according to the rate of progress and the resistance of the tissues; and the symptoms are dyspnœa, cough, expectoration, and hæmoptysis. The fungus does not spread to other parts of the body, and recovery may take place spontaneously.

CANCER AND OTHER TUMOURS OF THE LUNG

Cancer of the lung may be either primary or secondary. The latter is more common, and occurs mostly in the form of nodules scattered irregularly through the substance of the organ, or forming plates covering the surface of the pleura. The original seat of the growth may have been in the breast, the liver, or stomach, or one of the limbs. The lung is also sometimes invaded directly from the mediastinal glands, or by an epithelioma of the œsophagus. A true primary cancer of the parenchyma of the lung is quite rare, and the disease that has gone by this name has generally spread from the bronchial glands or the bronchial mucous membrane or the peri-bronchial tissue. It commonly invades the lung at the root, and may spread thence into the pulmonary substance, chiefly following the course of the branching bronchi. The organ may thus be largely converted into a mass of new growth. But before any great size is attained other important changes may occur. Thus the cancer grows into the lumen of the bronchial tubes, and by obstructing it causes bronchiectasis; or the growth breaks down into a granular detritus, and adjacent portions of the lung may become pneumonic or gangrenous; or by pressure on vessels a pleural effusion is caused, which compresses the lung. The cancer is more often of the softer or medullary variety.

Symptoms.—These vary with the position and distribution of the cancer growths—that is, according as they are seated in the main bronchus or bronchi, or are disseminated throughout the lung, or form one continuous mass or infiltration, involving a large portion of the organ.

1. Cancer involving the bronchus usually causes obstruction, the symptoms of which have already been described (*see* p. 498). Sometimes primary cancer of the mucous membrane causes death by profuse hæmorrhage.

2. When the lung is the seat of numerous nodules of growth, scattered indiscriminately through it, the patient suffers at first, at any rate, but little discomfort, and the physical signs are not very distinctive. Percussion resonance is normal, and the only change that may be observed is a diminution of the respiratory murmur all over the chest. In other cases the cancer nodules are more numerous, or set up bronchitic changes in the neighbouring bronchi; and more decided, though scarcely characteristic, symptoms may be produced. These are dyspnœa, very rapid breathing, lividity, frequent cough, and mucous expectoration; and on auscultation numerous rhonchi and râles are heard over the whole chest. The condition bears some resemblance to miliary tuberculosis, but the temperature may be normal. In other cases pleuritic friction sounds are heard in patches scattered widely over the lung.

3. Uniform infiltration of the lung is insidious in its course, and produces cough, dyspnœa, and expectoration of mucus, which is

sometimes tinged with blood, and sometimes mixed with larger quantities; occasionally it has a dark colour, and resembles currant jelly; but hæmoptysis is not very common. Pain is not generally a prominent symptom. The physical signs are those which must result from the infiltration of the lung with a solid material, at the same time that the bronchial tubes are filled up or blocked by the new growth. There is dulness, with absence or deficiency of breath-sounds, of vocal resonance, and of tactile vocal fremitus. There is thus a general resemblance to pleural effusion, which is often very deceptive. If the growth is considerable, the resemblance is increased by expansion of the chest-wall and displacement of the mediastinum: but a simple infiltration of the lung without large masses may lead to contraction of the side affected, and resembles rather cirrhosis or chronic pneumonia, or phthisis, or chronic pleurisy with partial absorption of fluid. Exceptionally, from the breaking down of the cancerous material, or as a result of bronchial obstruction and bronchiectasis, cavities are formed which produce characteristic physical signs. Sometimes the growth is accompanied by pleuritic effusion; this may be a purely serous liquid, or it may contain blood from rupture of the vessels of the new growth. The bronchial, cervical, and axillary glands become enlarged; and extension to the mediastinum may lead to symptoms of pressure, such as œdema of the head, neck, chest, and upper extremities, abductor paralysis of the vocal cords from pressure on the recurrent laryngeal nerves, obstruction of the trachea or one bronchus, or dysphagia from pressure on the œsophagus.

Cancer of the lungs, no less than that of other organs, is accompanied by progressive emaciation and loss of strength, and ultimately, in the course of from six to twelve months, death takes place, generally from exhaustion. The temperature is often normal, but it may be pyrexial. Sometimes, but not always, this is explained by an accompanying septic process in the bronchus or the pleura. (*See* Mediastinal New Growths.)

Diagnosis.—When cancer is known to exist in other organs, or when a cancer of the breast or of the jaw has been removed by operation, the presence of unaccountable dyspnœa should make one think of its occurrence in the lung; and in cases where the pulmonary symptoms are most prominent, the presence of large hard glands in the neck, or a tumour of the testis, or a rigid spine from implication of the vertebræ, may sometimes give the required clue. Extensive infiltration of the lung is most easily confounded with *pleuritic effusion;* and in elderly persons with the symptoms and physical signs of fluid, the possibility of cancer should not be forgotten. Exploration with the needle, or a trocar and cannula, or aspirator, may be necessary, and particles may then perhaps be obtained for microscopic examination. The sputum sometimes provides similar evidence. But if the exploration be negative, it may be that the needle has entered a lung collapsed from a cancer obstructing the root, and in this case bronchiectatic cavities may

afterwards develop, with offensive purulent sputum. On the other hand, if liquid be found, this does not exclude a cancer of the root of the lung; and this may well be suspected, if the liquid returns again and again after aspiration, while the temperature remains normal. Such a fluid should show, after centrifuging, only endothelial cells, and no leucocytes or lymphocytes (*see* p. 554). Blood-stained liquid, though occurring in cancer, is too frequent in other forms of pleurisy to be of much value in diagnosis. As implied above, a febrile temperature does not exclude cancer. The Röntgen rays may, of course, give valuable help.

The **Prognosis** is bad, and the **Treatment** must be confined to relieving pain and cough, procuring sleep, and supporting with good nourishing food. A liquid effusion accompanying the cancer may be aspirated, but it will probably return quickly.

Other forms of tumour occur in the lung, and are, as a rule, secondary to similar tumours elsewhere. Such are sarcoma, osteo-sarcoma, and enchondroma.

HYDATID OF THE LUNG

This parasite affects the lung in two ways. First, a cyst may form in the lung apart from, or even without, its occurrence in any other region; secondly, the lung may be invaded by a cyst in an adjacent organ rupturing through the parts which separate them. This is most common in hydatid of the liver.

A *primary hydatid of the lung* is very rare. It forms a globular cyst, with all the characteristics of the parasite as seen in the liver (*see* Hydatid of the Liver), but it is not generally surrounded by such a dense cyst of connective tissue. It is rather more frequent at the base than at the apex. It may be the only cyst in the body, or there are others at the same time in the liver, spleen, brain, or elsewhere.

Symptoms.—These depend upon its size, and upon the change it sets up in the surrounding lung. It may be so small as to yield no symptoms whatever. If larger, it must compress the lung-tissue, and it gives rise not uncommonly to hæmorrhages, and may cause pneumonia, or even gangrene. The patient has cough, dyspnœa, pain, and sometimes hæmoptysis. The physical signs, if any, are dulness, with more or less deficiency of vesicular murmur. If the cyst ruptures, and the contents are discharged by the bronchus, secondary cysts may be expectorated, which will at once reveal the nature of the case; afterwards the distinctive signs of cavity may be heard.

The lung may be *secondarily* invaded by *hydatids of the liver*. The pulmonary symptoms are then preceded by signs of hepatic disease, such as pain and tension in the hepatic region, some enlargement of the liver, and perhaps jaundice; and the earliest change in the lung is compression of its lower part by the enlarging cyst.

Increasing pain and distress, with more or less collapse or prostration, may mark the implication of the lung itself, and soon cough of paroxysmal nature is followed by the expectoration of bile-stained hydatid skins, or small and perfect cysts. More or less pneumonia, or even gangrene of the lung, may result, and thus the case may end fatally. But it is not uncommon for the inflammation of the lung to be limited in extent, and the whole of the hydatid may in time be expectorated through the bronchi, and the patient may thus recover completely.

Diagnosis.—A *primary* hydatid is most likely to be mistaken for phthisis, especially if the cyst is situated at the apex. A girl with the symptoms of a cerebral tumour had hæmoptysis, and was thought to have tubercle of the brain and pulmonary phthisis; but a hydatid cyst was found in the brain and another in the lung. The Röntgen rays should be used; and the patient's serum may be tested for specific anti-bodies (*see* Hydatid of the Liver). *Secondary* hydatid is generally recognised by the preceding hepatic trouble, and the appearance of bile and hydatid skins in the sputa.

Treatment.—No internal treatment can kill the parasites in the lung. If the diagnosis of a cyst sufficiently near the surface could be made with confidence, it might be treated by the surgical methods applicable in hydatid of the liver. Hydatid of the liver opening into the lung is commonly beyond the reach of surgical interference, and the treatment must be symptomatic, and in the main supporting.

SYPHILIS OF THE LUNG

Apart from the ulcerations of the bronchi, with resulting stenosis, which have been shown to be due to syphilis, the lung-tissue itself may exhibit the effects of the disease in various forms. One is that of the ordinary *gumma*, which is extremely rare in adults, though more common in infants, and gives rise to no recognisable clinical symptoms. Another is the so-called *white pneumonia* of syphilitic infants. The lungs are enlarged, white, dense, and firm; their section is smooth and opaque; they are sometimes resistant, at others easily broken down. The microscope shows a diffuse cellular inflammation of the lung, with thickening of the alveolar walls, and desquamation and fatty degeneration of the pulmonary epithelium. This condition may affect the whole lung, or one part may be uniformly altered, while the other contains only isolated areas. In another variety the alveoli are lined by cubical epithelium, and the connective tissue is replaced by a fibrous stroma infiltrated with cells from the cubical alveolar epithelium. Spirochætæ have been found in these cases. As these lesions are found chiefly in still-born children, they have but little clinical importance.

The extent to which syphilis may affect the lung in adults otherwise than by gumma has been the subject of much discussion. Destructive changes with bronchiectatic cavities may take place as

a result of bronchial or tracheal stenosis; and diffuse fibrosis, sometimes with cavities, has been found in syphilitic subjects, associated with marked pulmonary symptoms. The lesions occur especially at the root and central parts of the lungs, extending outward along the bronchi and vessels, and the cavities are often connected with obstructed bronchi. But these conditions are rare, and most cases of destructive disease of the lung occurring in syphilitic persons are due to tubercular disease.

DISEASES OF THE PLEURA

PLEURISY

Pleurisy, or inflammation of the pleural membrane, results in the effusion of lymph, or serous or purulent liquids.

Ætiology.—Some of the causes of pleurisy are easily recognised, such for instance as injury by fractured ribs: and the extension to the pleural surface of (1) lesions in the lung, like those of pneumonia, phthisis, pyæmic abscesses, cancer, tubercle, or hæmorrhagic infarcts; (2) lesions of the parietes, such as abscesses in the axilla, breast, neck, or abdominal cavity. The element of infection is obvious in most of these instances; and of the many cases which are apparently spontaneous, or are preceded by exposure to cold, a very large proportion are undoubtedly tubercular. Many have a history of tubercle, or they afterwards die of phthisis or other tubercular lesions. In many cases also the fluid inoculated into animals produces tubercular disease. Other infective agents causing pleurisy are those of scarlatina, measles, rheumatic fever, and pyæmia; and it is a frequent complication of Bright's disease. Pleurisy, pericarditis, and peritonitis may occur together as the result of the same infection (*see* Polyorromenitis).

The micro-organisms usually found are the following: Pneumococcus, streptococcus, staphylococcus, bacillus tuberculosis and b. typhosus; more rarely Friedländer's bacillus, b. coli communis, b. diphtheriæ, and micrococcus tetragenes. They are often combined, as, for instance, pneumococcus or tubercle-bacillus with streptococci or staphylococci; the last are not commonly found alone. In the sero-fibrinous effusions of tubercular pleurisies, tubercle-bacilli are often absent; they are more often present in tubercular purulent effusions. In purulent effusions of children, pneumococci are mostly found (80 per cent.), and in those of adults streptococci are more common (75 per cent.).

Morbid Anatomy.—The first stage of pleurisy consists of dilatation of the vessels of the pleura, quickly followed by exudation of the white corpuscles and fibrin on the free surface. Thus the membrane is at first minutely injected, but in the earliest visible

stage the naturally shining surface is rendered dull by the fibrin, which can be detached as an extremely delicate membrane. If the exuded material is more abundant, it forms thick layers, firm or pasty, generally rough on the surface, or villous, or reticular. Pleurisy may go no further than the formation of fibrin on the surface, and is then called "dry"; more often the fibrin is soon followed by the exudation of a serous or sero-fibrinous fluid, which may accumulate to the extent of two or three pints or more in the pleural cavity. This fluid has a yellow or greenish-yellow colour, a specific gravity of 1005 to 1030, often 1015 to 1018, and it becomes almost solid on boiling, from the albumin it contains. Not infrequently there are a few flakes of fibrin, or a quantity is deposited from the liquid a short time after its removal. The liquid is quite clear, or it is opalescent or turbid from the presence of corpuscles. In other cases the corpuscles are in sufficient quantity to form a thick layer at the bottom of the fluid after its removal, and there is every gradation between this and the formation of thick pus. Sometimes the liquid is more or less tinged with blood, proceeding from new-formed vessels in the false membranes.

This effusion of fluid is one of the most important results of pleurisy. Confined within the cavity of the pleura, it must displace the lung from its relations to the diaphragm and the wall of the chest, and in proportion as more fluid is effused, the lung becomes collapsed. This is not at first due to the actual pressure of the fluid, but to the elasticity of the lung, which naturally favours its retraction; and, indeed, it may be found that even a considerable quantity of fluid in the chest may fail to escape, or escape but slowly, on puncture, being held in, as it were, by the natural retraction of the lung towards the mediastinum: in other words, the pressure in the fluid is negative. But with a larger quantity a point is reached beyond the elastic collapse of the lung, the pressure in the fluid becomes positive, and the lung and surrounding parts are compressed, and displaced in various directions, in consequence. An important difference in the effects upon the lung is that the larger quantities of liquid compress the bronchial tubes, whereas with less amounts only the air-vesicles are deprived of air; and this explains some differences in the physical signs. The pressure which forces the lung towards the mediastinum pushes the mediastinum itself, with the heart and great vessels, towards the opposite side, bulges the wall of the thorax outwards, distends the intercostal spaces, and forces downwards the diaphragm with the subjacent liver or spleen.

Dr. Geddes found *post mortem* in a right-sided effusion that the mediastinum was more or less fixed by its attachments above and below, and the maximum displacement was at the level of the roots of the lungs. The heart was accordingly rotated so as to bring the apex downwards and slightly inwards. The right dome of the diaphragm was convex downwards; the right lobe of the liver was concave above, and depressed into the abdomen; and the left lobe sloped

downwards, while the central part was held up by the diaphragmatic attachments, falciform ligament, and hepatic veins. Over this fixed angle the right ventricular border of the heart was bent. The right auricle and ventricle were compressed, and the inferior vena cava was kinked.

In many cases pleurisy terminates by absorption of the effused products. The liquid disappears in the course of days or weeks, and the lung and the chest-wall finally come into contact either by expansion of the former, or by a gradual sinking in of the latter, or by a combination of these processes. The layers of fibrin covering the two surfaces have already probably become partly organised by the growth of new vessels from the pleura, and the formation of fibrous tissue; and, uniting together, they form in time a permanent layer of *adhesion* between the lung and the chest-wall.

A purulent pleurisy, usually known as *empyema*, appears sometimes to arise out of a serous pleurisy, or *simple effusion;* but it is often found soon after the onset of the symptoms: and is then undoubtedly primary. The early occurrence of empyema is most common in association with acute lobar pneumonia, in pyæmic and septic cases, after scarlatina, and as a result of perforation of the pleura from the lung or the abdomen. Its termination is by no means so favourable as that of simple effusion. Occasionally, no doubt, absorption takes place—that is, the fluid is taken up, the pus corpuscles become granular and fatty, and a caseous mass remains behind; or calcareous salts may be deposited in the residue. Sometimes an empyema finds its way through the pleural sac, either perforating the lung, so that the pus is expectorated; or "pointing" in one of the intercostal spaces, often the fifth, and bursting spontaneously. In either case air may find its way into the pleural cavity, and give rise to *pyo-pneumothorax*. Rarely an empyema opens through or behind the diaphragm into the abdomen. But, if unrecognised or untreated, it may remain a long time without perforating, with incomplete absorption, rendering the patient cachectic, and preparing the way for lardaceous degeneration of the viscera.

Both in serous and purulent effusions, the cavity is occasionally divided into separate spaces by adhesions between the lungs and the parietes. The fluid is then said to be *loculated;* and the condition is of importance when the case is treated surgically.

Symptoms and Physical Signs. *First Stage.*—The onset of pleurisy is characterised by a chill or rigor, with severe pain in the side, caused or aggravated by the act of breathing. In the apparently spontaneous cases, the pain is at first commonly at the side of the chest, or over the lower ribs; but in pleurisy determined by other lesions—as, for instance, phthisis—it may be situated elsewhere. The pain is cutting or tearing, and is intensified not only by breathing, but by coughing, sneezing, and every kind of exertion. The patient generally lies on his back or on the healthy side. There is mostly some pyrexia, in which the temperature may

reach 103°, but is more often 101° or 102°. With it are the usual accompaniments, furred tongue, loss of appetite, and malaise.

On examining the chest some impairment of movement on the affected side and deficiency of vesicular murmur at the painful spot are observed; but the characteristic physical sign is the *pleuritic rub*, or friction sound, which arises by the movement upon one another of the two pleural surfaces, roughened by exudation (*see* p. 453). The sound varies with the degree of friction, and this may be so great that it can be felt by the hand placed on the chest, as well as heard with the stethoscope: on the other hand, there may be no rub in this first stage, if the patient is prevented by the intense pain from making the inspiratory movement necessary to produce it.

Stage of Effusion.—When liquid is effused, the two pleural surfaces become separated, the friction sound disappears, the pain diminishes, and symptoms and physical signs occur which are the direct result of the presence of liquid and the compression or displacement of the various organs which it effects. The chief symptom is shortness of breath, especially on exertion, and this dyspnœa is in proportion to the amount of liquid effused. It is often scarcely observed when the patient is still, but becomes manifest when he moves about, or even when he talks. He lies on his back, or on the affected side, to allow the greatest freedom to the healthy lung. He may be entirely free from cough, or may have slight cough without expectoration. Fever commonly persists as before, the temperature ranging from 100° to 103°. The pupils are often unequal in pleuritic effusion, that on the affected side being larger.

As the fluid gravitates to the most dependent part of the chest, small quantities are usually detected at the base behind, where there is absolute dulness, while vesicular murmur, vocal resonance, and tactile vocal fremitus are much enfeebled, or entirely absent. With a considerable quantity of fluid the following physical signs are observed: On inspection, the affected side of the chest is motionless, and may be obviously larger than the other; the intercostal spaces, instead of being slightly depressed below the level of the ribs, are filled up (or "obliterated"). The heart is displaced: with effusion on the right side, its impulse may be perceived beneath or outside the left nipple; with effusion on the left side, an impulse is often felt in the intercostal spaces to the right of the sternum, generally the third, fourth, and fifth, even as far as the right nipple, and in rare instances beyond it. The dulness in such cases is observed in front, in the axilla, and behind, and is continuous with dulness on the opposite side corresponding to the displaced heart. The upper margin of the dull area is horizontal, but near the spine behind it falls an inch or more, so that it forms a convex curve upwards (*Damoiseau's line*). At the same time on the opposite side of the chest the resonance is not unaffected, for a triangular area of dulness is found of which the apex is close to the spine at the level of the curve above described, and the base extends from

the spine along the twelfth rib for from two to three inches (*Grocco's paravertebral triangle*). The dulness diminishes in this area when the patient lies on the side of the effusion (*see* Fig. 48). The cause of Grocco's triangle has been much discussed. Ewart's view that the pressure of the pleural fluid limits the vibrations of

FIG. 48

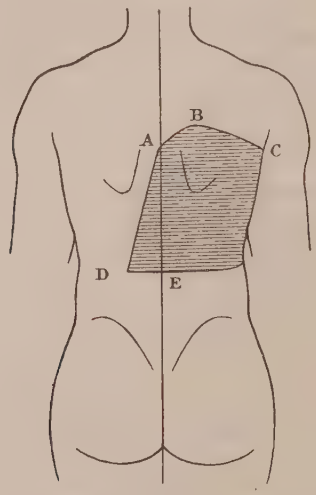


Diagram illustrating Percussion posteriorly with Pleural Effusion on the right side.

A B C, Damoiseau's line. A D E, Grocco's triangle.

the vertebræ and adjacent heads of the ribs on the healthy side seems plausible.

The liver on the right side, or the spleen on the left, may be pushed down; and the descent of the diaphragm on the left side leads to dulness at the upper part of that space between the left lobe of the liver and the spleen, which normally yields gastric resonance (*Traube's space*). When the liquid is sufficient only to reach about two-thirds the height of the chest, there may be heard the peculiar modification of the percussion note under the clavicle and above the level of dulness, which is known as *Skodaic resonance* (p. 450); and sometimes heavy percussion elicits a sound closely resembling the cracked-pot sound of a phthisical cavity.

Over the dull area there is diminution or absence of breath-sounds, of vocal resonance, and of tactile vibration. Of these, the last is very constant, but the breath-sounds, instead of being absent, are sometimes bronchial, especially at the upper part of the area of dulness; and this is to be explained by the bronchial tubes at this level being still patent, although the vesicular tissue is collapsed and the lung inactive. The conditions for the production

of bronchial breathing are thus the same as in pneumonic consolidation. Generally, however, the bronchial breathing is less loud and ringing—softer, or more distant, and often of higher pitch. Occasionally, especially in children, the bronchial breathing is heard over the whole area of dulness. Where the breath-sounds are bronchial, the vocal resonance is often increased. It is especially in pleuritic effusion that the modification known as *ægophony* is heard (p. 450). Bronchial breathing and bronchophony may be heard over the area of Skodaic resonance. On the opposite side the breath-sounds are exaggerated, but over Grocco's triangle they are diminished.

In extreme cases the whole of one chest is dull—back, front, and side, and from apex to base. Generally, bronchial breathing is entirely suppressed, except along the spine at the root of the lung, and ægophony is less frequently present than in more moderate degrees of effusion. The mediastinum and heart are displaced, and the diaphragm is depressed on the same side. The disturbance of the respiratory functions may at length be fatal. The patient becomes more and more livid, rhonchi and mucous râles are heard in the hitherto healthy lung, and asphyxia ensues. Sometimes there is sudden syncope, which may reasonably be explained by the compression of the cardiac cavities and the kinking of the inferior vena cava already described (*see* p. 548); when a very slight additional disturbance may completely stop the circulation at this point.

Very rarely the pulsation of the heart (or perhaps the aorta) is communicated to a pleural effusion, either as a shock or wave transmitted to a large serous collection, or as a more localised, perhaps visible, pulsation in an empyema, pointing through the chest-wall. This is described as *pulsating empyema* or *pulsating pleurisy*.

Progress to Recovery.—Where recovery takes place spontaneously the liquid disappears, sometimes gradually, at others with remarkable quickness. The upper parts of the area of dulness become resonant, the vesicular murmur returns, and often with it the friction sound is again heard, louder, longer, and over a much more extensive surface than was the case previous to the effusion. In addition, the rub is more distinctly palpable, but much less painful. While the liquid is being absorbed, and the lung is beginning to expand, the chest often falls in—a change which is first observed behind, where the naturally round chest becomes flat as compared with the other side. In extreme cases the chest is flattened in front and behind, these two surfaces meeting in a rounded angle at the axilla; at the same time, the dorsal spine is curved, with the concavity towards the affected side, the shoulder is depressed, and the angle of the scapula projects from the ribs. Frequently the physical signs at the base remain abnormal for some time, suggesting the continuance of liquid; but this is undoubtedly due to incomplete expansion of the extreme base, with or without a layer of new fibrous tissue (thickened pleura), the result of the inflammation.

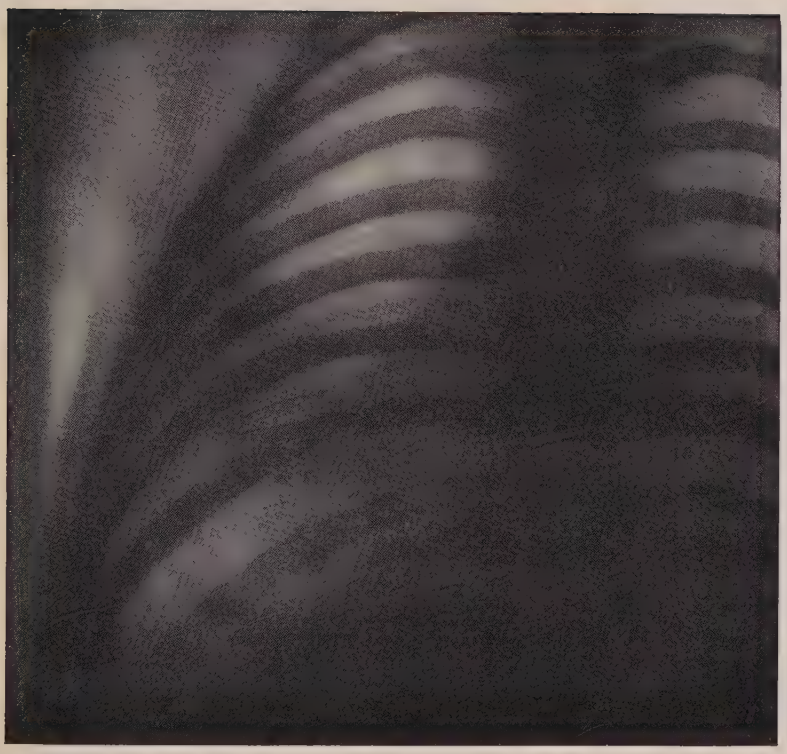
In some cases a chronic pleural effusion results, the liquid neither increasing to a fatal result nor becoming absorbed.

Diagnosis.—In the early stages the pain of pleurisy has to be distinguished from other pains in the chest, the most common of which is *pleurodynia*, or muscular rheumatism. This is increased by movement, but is unaccompanied by fever or by rub. Intercostal *neuralgia* is distinguished by its relation to the nerves, and by the tender points characteristic of neuralgia. Affections of the liver, spleen, or colon may give rise to pains aggravated by breathing, for these structures are compressed by the descent of the diaphragm during inspiration.

In the second stage we have to consider, first, whether there is liquid in the pleural cavity; and secondly, what is the nature of the liquid, whether serum or pus. In acute cases, pleurisy and *pneumonia* are most likely to be confounded together; and it must especially be remembered that bronchial breathing may occur in both. Pleurisy is generally characterised by the absence of tactile vibration and the more absolute percussion dulness; and the larger effusions cause displacements of the heart and of the diaphragm, which are conclusive; Grocco's triangle is not observed in pneumonic consolidation. Generally, also, pleurisy is wanting in the very high fever, pungent heat of skin, and flushed face of pneumonia. The two may occur together; and then, if the pleural effusion is sufficient to compress the lung, the bronchial breathing characteristic of pneumonia will be modified or absent; and the negative signs indicative of the lung being compressed by fluid will predominate. But, should the sputum be rusty, pneumonia is certainly present. With the Röntgen rays a very dark shadow, whose upper margin rises sharply from the spine towards the axilla, is produced by pleural effusion; and if the effusion is large, the lines of the diaphragm and the lower ribs are not seen. The lung above shows a faint darkening from condensation; and a displacement of the heart is generally obvious (*see* Plates III. and IV.). In pneumonia there is often a light space at the extreme base, and the outline of the heart is less displaced than in pleural effusions. The early signs of pleurisy are sometimes so little marked, and the effusion of liquid is so insidious, that the local features of the illness may be overlooked, and the patient may be thought to be suffering from *typhoid* or other form of fever.

In chronic cases, fluid may be simulated by most *consolidations* of the lung-tissue, whether from deposit in its substance or from external compression; such are some cases of tubercular consolidation, the induration which results from heart disease, cancerous growth in the lung, compression from the front by pericardial effusion, and from below by subphrenic abscess, cancer or hydatid of the liver, cancer or hydatid of the spleen, and abscess about the spleen or kidneys. The physical signs common to these conditions are dulness, loss of breath-sound, of vocal resonance, and of tactile vibration—that is, absence of all evidence of healthy lung—simply

PLATE III



Skiagram of a small pleuritic effusion on the left side in a child of four, showing a dark area at the base, with curved upper margin, sloping upward from spine to axilla, and a light area below, due to air in the stomach. The heart is not displaced. (Taken by Dr. A. C. Jordan.)

[*To face p.* 552.

because they compress or push up the lung in the same way as liquid effusion does, and do away with that spongy tissue in communication with the trachea and larynx, upon which the normal physical signs depend; but with less degrees of compression the breath-sounds are here also sometimes bronchial. Most of these changes affect the base of the lung, and no help can be gained from the absence of the signs (enlargement of chest or displacement of heart) which occur in the more abundant effusions. Cancer of the lung is especially deceptive, because it may form a tumour, occupying and enlarging one side of the chest, and pushing the mediastinum and heart to the other side. It is more common in those over middle age, and diagnosis may be impossible without the aid of an exploration. In tumours, abscesses, or cysts rising from the abdomen, the upper limit of dulness in the axilla may form a curved line dome-shaped or convex upwards, instead of the horizontal line common in pleural effusions. If on the left side the gastric resonance near the left costal margin (*Traube's space*) is encroached upon by dulness, there must be something more than pneumonic consolidation; but even a large liquid effusion does not always reduce this area of resonance.

It is always desirable to know whether the effusion is serous or purulent: the only certain way is to withdraw some fluid from the chest by a suitable exploring needle and syringe. The duration of the symptoms is no guide; but effusions are especially likely to be purulent when they have followed the eruptive fevers, such as scarlatina, or occur in connection with acute pneumonia or with phthisis. Baccelli's sign, the better transmission of the voice through serum than through pus, cannot be relied on. Slight flattening of the chest, showing that some absorption is taking place, suggests, but is not conclusive in favour of, serum.

Help is often gained from the general conditions which accompany pus in the chest as well as elsewhere. Thus a sallow appearance or even marked anæmia may be present; the temperature has often a hectic type, ranging from 98° or 99° in the morning to 102°, 103°, or 104° in the evening, and with this rigors or profuse sweatings may occur. But serous pleurisy may be accompanied by high fever and sometimes free sweating; and, conversely, the chest may be full of pus when the temperature is quite normal. There may be a slight leucocytosis even in serous pleurisy, though it is often absent when the pleurisy is tubercular: but there is pronounced leucocytosis in empyema.

The sudden occurrence of purulent expectoration, especially if offensive, in the course of pleurisy, is an important indication of empyema; and in cases of long duration the ends of the fingers become thickened, or "clubbed." Œdema of the chest-wall occurs more frequently in empyema than in serous effusions; but in neither case is it an early sign.

Serum may be present in the pleural cavity from other causes than pleurisy—namely, local and general dropsy. The physical

signs may be the same, but *hydrothorax*, as the condition is called, generally follows upon disease of the heart, or Bright's disease, or pressure upon vessels by cancer in the chest; and there is an absence of the febrile accompaniments of pleurisy.

The method of *cyto-diagnosis* may help to a knowledge of the origin of the effusion. The liquid is centrifuged, and the sediment of cell elements is stained and examined microscopically. In passive effusions (hydrothorax) large endothelial cells predominate; in infective forms of pleurisy due to streptococcus, pneumococcus, or typhoid bacillus, the polymorphonuclear and large uninuclear leucocytes are found in excess; while in tubercular pleurisies, the predominant elements are lymphocytes with some red blood-corpuscles. A blood-stained serum is not distinctive of any one pathological condition.

Inoculation of animals with pleuritic liquids will show their tubercular origin, when to culture methods they appear to be sterile.

Prognosis.—Most cases of pleurisy without effusion, or with a sero-fibrinous effusion, get well, either under medicinal treatment or after surgical evacuation of the liquid; but their subsequent history is often unfavourable (*see* p. 545). Empyema is more amenable to treatment in children than in adults, and more promising the earlier the pus is evacuated. This is probably because in children the majority of cases are pneumococcal, while in adults more are streptococcal (*see* p. 545). If a pneumococcal empyema is complicated by pericarditis, which is not infrequently the case, the prognosis is bad; but cases have recovered.

Treatment.—In the treatment the first consideration is the pain in the side. This may be generally met by the application of linseed-meal poultices and the administration of opium or morphia internally, or morphia subcutaneously. The use of blisters, of leeches, or of cupping over the painful spot, also generally gives relief. The affected side may be strapped, and by this means the respiratory movements are restrained, the pain is allayed, and inflammatory action is probably in some measure checked. The strapping should be applied in broad strips from the spine to the sternum, alternate strips passing obliquely upwards and obliquely downwards, till the whole side is covered. The patient should be kept at rest, and placed on a diet suitable to his febrile condition. If effusion takes place, anodynes will be less needful, and salines, such as the acetate and citrate of potassium or ammonium, should be given, partly as refrigerants, partly for their effect upon the excretions of the skin and kidney, the increase of which will favour absorption of the effused fluid. After a time iodide of potassium, squills, or other diuretics may be added, and absorption may be encouraged by the application of the tincture or solution of iodine over the affected side.

Good results are said to ensue from *auto-sero-therapy*, of which the procedure is as follows: A sterilised syringe and needle are used

PLATE IV

Skiagram of pleuritic effusion on the right side, showing dark area with upper margin sloping steeply upward from spine to axilla. (Taken by Dr. A. C. Jordan.)

[*To face p.* 554.

to draw 4 or 5 c.c. of liquid from the pleura; the needle is then drawn out from the pleura and intercostal muscles and pushed at once into the subcutaneous tissue. Two c.c. of the fluid (if serum or blood) are then injected under the skin, and the needle is withdrawn.

But in most instances the actual removal of the liquid by puncture and aspiration becomes desirable. There are three cases in which this should certainly be done: (1) When the effusion fills the whole of one chest; (2) when the liquid, though not so abundant, has been present two or more weeks, and in spite of treatment shows no sign of being absorbed; (3) when the liquid is purulent, whatever its quantity may be. Some difference of practice obtains as to removal in the early period of an effusion, since it is quite possible that absorption may take place without it, and for a time at least the effusion may be regarded as giving rest to the inflamed surfaces by separating them. But there are many arguments on the other side. We have little, if any, means of knowing how long an effusion may last if we do not interfere; as a rule, the longer the liquid is present, the more difficult it is for the lung to expand, both from changes in its substance and from the deposit of fibrin in its surface; the febrile temperature often falls directly the fluid is removed, even though it be only serum; and in many cases relief is at once afforded, and convalescence is uninterrupted. Thus even a moderate quantity of liquid should be removed after a fortnight, if it is not by that time distinctly diminishing.

The liquid may be evacuated by the aspirator (Potain's or Dieulafoy's), which should be perfectly clean and aseptic. The site chosen for puncture may be the seventh or eighth space in the posterior axillary line, but when there is not much liquid it may require a puncture in the ninth space, near the angles of the ribs. I believe that it is best, as a rule, to remove as much liquid as will come. When, during aspiration, the end of the fluid has been nearly reached, the patient often suffers pain at the seat of the puncture, and he begins to cough, probably as a result of the admission of air to the newly opened bronchial tubes. Blood may now appear in a fluid previously bloodless; and this should, as a rule, be an indication to stop the operation, withdraw the needle and close the wound. Resonance generally returns at the upper part of the chest at once, but, in spite of the withdrawal of three or four pints from the chest of a grown-up man, the physical signs may persist for some time at the base, in consequence of the fact that though the liquid has been removed, the lung remains collapsed. If the liquid does not reaccumulate the temperature will subside, and the patient will feel an increasing improvement in his capacity to breathe. If the pyrexia and dyspnœa persist, and the physical signs extend, the chest may be again explored.

Syphonage is preferred by some to the aspirator—that is, after puncture the liquid is drained through a flexible tube into a vessel placed on the floor.

If an exploration by the needle shows that the liquid is purulent (empyema), the surgeon should make a free incision, under an anæsthetic, local or general, into the eighth or seventh space, with the usual antiseptic precautions, insert a large red rubber tube, and allow the pus to drain into antiseptic dressings. In some cases, in spite of free drainage and thorough antiseptic treatment, the cavity continues to secrete pus, and the wound does not close. If this goes on too long the supervention of lardaceous disease is to be feared, and further effort should be made by removing a portion (1½ to 2 inches) of a rib or ribs adjacent to the wound. This widens the aperture for drainage, and allows the bony thorax to fall in upon the imperfectly expanded lung. When the ribs are very close together, resection may sometimes be done with advantage at the first operation. An empyema will sometimes be cured by a single aspiration, and this appears likely to occur, especially when the pus is due to pneumococci, whose virulence is short-lived; but I believe time is saved by early incision. During the surgical treatment of an empyema the patient should be supported in every way by good food, stimulants, fresh, bracing air, and by tonic medicines, such as quinine, iron, and cod-liver oil.

HYDROTHORAX

This term is applied to the collection of fluid in the pleural cavity, not as a result of inflammation, but in consequence of heart disease, or Bright's disease, or interference with the circulation in the chest by cancer or tumour. It is, indeed, dropsy of the pleural cavity; and the liquid contains less albumin and less fibrinogen than are found in pleurisy. Its physical signs are similar to those of pleuritic effusion, but the rub is, of course, absent. Arising, as it often does, from a general or central cause, it is much more often bilateral than pleurisy is; but occasionally a very large one-sided effusion may be merely dropsy. It is also stated, as another point of distinction, that when the position of the body is changed from recumbent to erect, or *vice versâ*, the line between dulness and resonance (that is, the upper level of the liquid) is likewise altered, rising higher in front when the patient sits up; whereas this mobility of the liquid is scarcely, if at all, noticeable in pleuritic effusions, which are confined to one situation by the surrounding adhesions. The recognition of hydrothorax, however, generally depends on the history and the previous existence of the diseases which cause it. When the liquid has been removed, the kind of cellular elements it contains may help the diagnosis (*see* p. 554).

Its Treatment is mostly of secondary importance, being involved in that of the cause. As the liquid is almost certain to recur if removed, paracentesis or aspiration should only be performed where a very large effusion, whether on one side alone, or divided between the two, is seriously impeding respiration.

HÆMOTHORAX

By this term is meant the effusion of blood in quantity into the pleural cavity: it is not used for the merely blood-stained serous effusions so common in pleurisy. Hæmothorax commonly results from injuries or from rupture of a thoracic aneurysm. It occurs sometimes in tuberculosis, either from tubercular disease of the pleura, or from rupture of a pulmonary vessel into a cavity and later extravasation into the pleura. Exceptionally it occurs from bursting of an emphysematous bulla (Newton Pitt); or from degenerated vessels in association with cirrhosis of the liver, granular kidney, or dilated heart; or from malignant disease. And sometimes it appears to be primary and the origin is never explained. The **Physical Signs** are those of liquid in the pleural cavity. The **Diagnosis** will depend, in the case of aneurysm, on the previous history, and on syncope and pallor indicating rapid loss of blood. It may only be discovered on exploration.

Treatment.—If the liquid be aspirated it is very likely to return; and probably it is better to leave the blood to be absorbed, unless it is threatening life by direct pressure.

PNEUMOTHORAX

Pathology.—The presence of air in the pleural cavity constitutes a *pneumothorax*. If serum is present at the same time it is a *hydro-pneumothorax;* if pus accompanies the air, a *pyo-pneumothorax;* if blood, a *hæmo-pneumothorax*.

A pneumothorax may be brought about by any wound in the side which passes through the whole thickness of the chest-wall, and it is often produced by a fractured rib puncturing both layers of the pleura, so as to let out air from the lung into the pleural cavity, while the skin remains intact. Much more often, pneumothorax is the result of disease, and especially of phthisis, from the rupture or sloughing of the pleura over a vomica; and less commonly an empyema makes its way through the pleura into the lung, and air passes into the pleural sac, so as to form a pyo-pneumothorax. Similarly, pyo-pneumothorax occurs in the surgical treatment of empyema by incision. Rarely in acute pneumonia the pleura ruptures, and air escapes; or a pyæmic abscess or gangrene may lead to a similar result; or an emphysematous bulla may burst. Air may also enter the pleura in consequence of spinal or mediastinal abscess, burrowing into the pleura; and ulcer or cancer of the stomach, or cancer of the œsophagus, may let in air from the alimentary canal.

A pneumothorax may be described as open, closed, or valvular, according to the condition of the opening which created it.

Open Pneumothorax.—When air enters the chest from an external

wound, and the wound remains patent, the lung collapses by its own elasticity; and not only the lung of the wounded side, but also the opposite lung, contracts somewhat and draws with it the mediastinum, so that lateral displacement of the viscera takes place, just as it does in liquid effusion. The same happens if the pneumothorax results from rupture of a cavity in phthisis, supposing the aperture to remain patent, so as to keep the pleural sac in communication with the bronchial tubes. In both these cases the mean pressure of the air in the chest is equal to that of the atmosphere.

Closed Pneumothorax.—When the aperture is small it may be quickly closed by lymph; further extravasation is prevented, and the air may then be completely absorbed. This happens in cases of laceration of the pleura by fractured rib, and sometimes, or to a less complete extent, in pneumothorax from disease of the lung. It can be understood that the conditions are much more favourable to absorption in pneumothorax from injury than in that from phthisis. In closed pneumothorax, the pressure has been found to be negative, and the displacement is, *cæteris paribus*, less than in the first case.

Valvular Pneumothorax.—A third possibility is that a shred of pleural membrane or lymph hangs over the aperture, so as to form a valve. The air is then drawn into the pleural sac by inspiration, but is unable to escape during expiration; the mean pressure becomes positive—that is, it exceeds the pressure of the atmosphere, and the displacement of viscera and distension of the chest may be extreme; thus, the heart may be pushed far over to the opposite side, and the liver or spleen may be driven down by the flattening or inversion of the diaphragm. A valvular opening may, like others, become closed by adhesions.

The amount of collapse of the lung, and displacement of the viscera, is influenced in different cases by the previous condition of the lungs. This is a point of importance from the large proportion of cases (nine out of ten) occurring in phthisis. If the lung is extensively diseased, or in great part adherent, the collapse will be less than if the lung, for the most part healthy, has only a small amount of disease, with cavity, at the apex. The entrance of the air into the pleural cavity may be otherwise harmless, especially in traumatic cases; but a serous or purulent effusion is commonly the result of the entrance of micro-organisms, especially when phthisis or a pyæmic abscess is the antecedent. If the effusion is serous, it may be absorbed together with the air, but a pyo-pneumothorax commonly persists unless dealt with surgically. The air of a pneumothorax differs from that of the atmosphere in containing very little oxygen, a large quantity of carbonic acid, and excess of nitrogen. Different analyses have shown in 100 parts—oxygen, from 2 to 5; carbonic acid, from 6 to 16; nitrogen, from 80 to 90.

Physical Signs.—Over the affected side there is marked hyper-resonance of tympanitic quality, changing, of course, to dulness at the lower part when liquid is present at the same time. It is

stated that in rare cases of extreme distension as the result of a valvular aperture, the percussion note may become muffled or actually dull. The respiratory murmur is often entirely inaudible, or a faint amphoric breathing is present. When this kind of breathing is loud or well marked, it is probably due to the aperture being patent; but a fainter sound may occur even when adhesions have shut off the lung from the pleural cavity. Vocal resonance and tactile vibration are generally much diminished, but bronchophony or pectoriloquy may be present. Sounds of a metallic or tinkling character are sometimes heard when the patient breathes, or speaks, or coughs; these are probably due to an echo of the vibrations by the side of the thorax. One characteristic sound is due to the dropping of fluid from the upper part of the chest into the liquid below, the noise being reverberated with almost musical quality. The *bruit d'airain* or *bell sound* (*see* p. 455) can also be elicited in cases of pneumothorax.

If there is also liquid effusion, its gravitation to the lowest part of the chest under all circumstances is well shown. If the patient is recumbent, the posterior part of the chest is dull, and the anterior part is tympanitic; if the patient now sits up, the lower part of the chest, back and front, becomes dull, while the upper part, back and front, is resonant. If *Hippocratic succussion* be employed, a splashing sound will be obtained (*see* p. 456).

The **Symptoms** of pneumothorax are very variable, depending largely upon the amount of antecedent disease. If it supervenes upon a lung extensively diseased, it may add but little to the distress already present; if it occurs in a lung for the most part, or entirely, sound, the symptoms will be pronounced; lastly, if in a case of phthisis with extensive disease on one side, pneumothorax occurs on the other side, the result may be quickly fatal. The symptoms in the severe cases are sudden pain, with a sense of something giving way internally, then distress of breathing, with more or less collapse, small pulse, lividity, and sweating. The breathing is shallow and rapid; the chest is distended on the affected side, and the intercostal spaces are depressed on inspiration.

These troubles may be aggravated until death takes place, within a few hours, or two or three days; or the first severe symptoms may subside, and comparative ease may follow, but generally with rapid breathing and orthopnœa.

Diagnosis.—*Emphysema* may be for a moment confounded with pneumothorax, but it is always bilateral unless compensatory on one side to disease on the other. A very large *cavity* in phthisis may sometimes simulate a localised pneumothorax in its hyper-resonance, feeble vesicular murmur, and tinkling sounds; but *bruit d'airain* must be rare in vomica, and the flattening of the chest over a cavity will generally serve to distinguish it from a pneumothorax. On the other hand, in some cases pneumothorax may be overlooked from the absence of any special symptoms at the time of its occurrence. Obstruction of a bronchus will cause the disappearance of

the vesicular murmur; and in the early stages of compression by an aneurysm there may be at the same time so much distension of the lung as to yield hyper-resonance, and thus give rise to a mistaken diagnosis of pneumothorax (*see* p. 499). *Ruptured diaphragm*, with escape of the stomach into the thorax, may closely resemble pneumothorax, especially as they may both arise from the same injury—a contusion of the chest; and it may also be simulated by an unusually high position of the stomach in the chest in consequence of contraction of the left lung, and by abscess beneath the diaphragm containing air (*subphrenic pneumothorax*). The Röntgen rays show the transparency due to air in the pleural cavity, the collapsed lung, the depressed diaphragm on the same side, and the displaced heart (*see* Plate V.).

Prognosis.—In many cases it is the final event of phthisis, and death takes place in a few days; but some patients live for weeks or months with hydro-pneumothorax. Complete recovery with absorption of the air may take place, as in cases due to injury, in those following strain in whooping-cough, or pneumonia, and very exceptionally even in phthisis.

Treatment.—This is, in the main, palliative. For the intense pain and distress accompanying the rupture, opium or a subcutaneous injection of morphia ($\frac{1}{3}$ to $\frac{1}{2}$ grain) should be administered, and hot poultices and fomentations should be frequently applied. Stimulants, as wine, brandy, or ether, may also be required. In cases of extreme distension it may be desirable to perform paracentesis, a trocar and cannula being inserted between the ribs over the resonant area and the air removed by syphon action; the relief is, as a rule, only temporary, and the paracentesis may have to be repeated. Aspiration is generally undesirable, because it may keep open the aperture in the lung, and may draw septic matter from the lung into the pleura. If the communication with the lung becomes closed, the air will probably be absorbed, and the serum may be removed by paracentesis. A pyo-pneumothorax should be treated, like an empyema, by incision and drainage.

Skiagram of a case of pneumothorax, showing collapsed lobes of the right lung and the heart displaced to the left. The right side of the chest is lighter than normal, being filled with air, and the right half of the diaphragm is depressed. The left lung is also somewhat light, and the diaphragm somewhat low, from compensatory distension. (Taken by Dr. A. C. Jordan.)

[*To face p.* 560.

DISEASES OF THE ORGANS OF CIRCULATION

Before describing the methods of investigation in diseases of the organs of circulation, some note must be made of recent work upon the structure and mode of action of the heart's muscle. The existence of cardiac nerves and ganglia, and the well-known effects of emotion upon the heart, as well as physiological experiments, make it clear that the nervous system is intimately concerned with the movements of the heart; but it is also certain that the muscular tissue has powers of contraction independent of any visible nerve structures. The myogenic theory of the heart's action receives a good deal of support (1) from recent discoveries in the anatomy of the heart, (2) from pathological changes in these structures, and (3) from observations on the action of the heart in various forms of disease. As is well known, after each ventricular systole there is a period of rest from contraction, which is broken by the systole of the auricle, and this is immediately followed by the systole of the ventricle. Thus, the auricle initiates the movement, which is then transmitted to the ventricle. Three structures appear to be involved in this process: one a small nodule of nerve tissue and muscular fibre situated near the attachment of the superior vena cava to the auricle, called the sino-auricular node; another, a similar nodule situate in the septum of the auricles, near the opening of the coronary sinus, called the auriculo-ventricular node; thirdly, a special band of muscular fibres known as the auriculo-ventricular bundle of His. This band arises in the auriculo-ventricular node and measures about 2·5 mm. in breadth: it passes from the auricular septum into the ventricular septum, lying at first below the *pars membranacea septi*, and there divides into two portions, which lie one on each side of the septum ventriculorum: each then is distributed extensively in the wall of its own ventricle, terminating in so-called Purkinje's fibres in one of the moderator bands. In the normal action of the heart, the stimulus appears to start in the sino-auricular node, and to be transmitted to the auricles, and from them by the bundle of His, to the ventricles. Experimental and pathological interference with these structures modifies the action of the heart. Gaskell by constricting the auriculo-ventricular ring in the tortoise's heart, caused the auricles and ventricles to beat independently of one another, the auricles beating faster than the ventricles. Erlanger succeeded in clamping the bundle of His in the heart of dogs and other animals, and by increasing degrees

of compression caused first an occasional failure of the ventricular contraction, then a ventricular beat only with every other auricular beat, or ventricle to auricle as 1 : 2; then as 1 : 3, and 1 : 4; and finally the ventricles beat quite independently of the auricles, implying complete interruption of the transmission of impulses from auricle to ventricle. This condition is now called *heart-block*, in the early stages partial, in the last condition complete. In the human heart a similar condition is produced by structural disease involving the bundle; and by toxic agents, of which digitalis is one, presumably operating in the same way. But much more frequently, in other circumstances than definite interference with this conducting band, various stimuli or irritations lead to disorderly cardiac contractions, which may be initiated either in the auricles, or in the ventricles themselves. By such are explained the intermissions, extra-systole, and other forms of irregular action which will be described.

Much work upon this subject has been done by means of synchronous observations upon the movements in the jugular vein, which represents the right auricle, and hence presumably the left auricle, and upon the radial pulse, which represents the ventricular movements; and such observations have been more fertile of results than the older records with the *cardiograph;* and there is much promise from the use of a new instrument, the string-galvanometer, by which *electro-cardiograms* are obtained. From such records inferences can be drawn as to the five functions now recognised to be possessed by the cardiac muscle, namely, excitability, contractility, rhythmicity, conductivity, and tonicity.

PHYSICAL EXAMINATION OF THE HEART AND VESSELS

Like the lungs, the heart is accessible to examination by the eye, the hand, and the ear. It comes into close proximity with the chest-wall between the anterior margins of the lungs, over an area corresponding to the lower half of the sternum on the left of the middle line, and the inner portions of the fourth and fifth left costal cartilages and the spaces below them. The *impulse* of the heart can be determined by inspection and palpation; the *præcordial area*, or the part of the heart exposed between the lungs, can be made out by percussion, and the *heart-sounds* can be studied by auscultation.

INSPECTION

In health, the heart can be commonly seen to beat in the fifth intercostal space from half an inch to one inch within a line drawn vertically down from the nipple, or from two and a half to three inches from the middle line in an average-sized adult; this is called

the *impulse*, or *apex-beat*. The former is the better term, because the point of visible impulse is not the actual apex of the heart, but is situate internal or to the right of it. The impulse is normally limited to an area of half an inch in diameter.

In disease, various changes take place in the position and the character of the impulse. It may be in the sixth or seventh intercostal space, or in the fourth or third: it may be in the nipple-line, outside it, or in the axilla: it may be much nearer to the sternum than usual. Sometimes the beat of the heart can only be seen to the right of the sternum; or the impulse may extend over two or three intercostal spaces from the fifth upwards; or, in addition to the impulse in the usual position there may be one below the ensiform cartilage (lower part of the right ventricle), or in the second left intercostal space (top of the right ventricle). Sometimes no impulse can be seen at all, either from feebleness of beat, or because the heart is overlaid by lung. In character the beat may be unusually forcible, or heaving, or quick, or irregular.

Inspection also shows bulging of the chest-wall in some cases of great enlargement of the heart.

Movements in the epigastrium are often produced by the heart's contractions. A slight *systolic retraction* is not uncommon in healthy persons: a more marked retraction occurs with hypertrophy of the heart. If the right ventricle is chiefly hypertrophied a *systolic impulse* may be produced; and one slightly later in time occurs from the impact of the aorta, whether aneurysmal or conducted by tumour, and by a pulsating liver.

Röntgen Rays.—By means of X-rays, the position, shape, size, and movements of the heart and great vessels can be seen and mapped out. The normal heart shows on its right border a convexity due to the right auricle, and above this a curve due to the great vessels: on its left border from above downwards are convexities representing the aorta, the pulmonary artery, the left auricle, and the left ventricle. The enlargement of any one of these parts in disease can be recognised by corresponding alterations in the outline.

PALPATION

Examination with the hand confirms much that can be seen with the eye as to the position and character of the impulse, but the hand is not equal to the eye in the appreciation of the finer movements in the intercostal spaces. Vibrations which correspond to sounds heard with the stethoscope (*see* Auscultation) are sometimes felt by the hand. Over the base of the heart, mostly in the second left intercostal space, the closure of the pulmonary valves, which forms part of the second sound, may be felt as a sharp, short click; and in certain cases of valvular disease a *thrill* can be felt over a limited area, where the stethoscope reveals an abnormal sound, *bruit*, or *murmur*. Such a thrill may in different cases be felt at nearly all the orifices of the heart, but the most frequent is that

which is presystolic in time, and is felt near the apex of the heart in mitral stenosis. Aneurysms, perforation of the septum, and pericardial lymph also give rise to palpable vibrations.

PERCUSSION

While the greater part of the chest is resonant to percussion, from the presence of the lung, there is a small area over the surface of the heart which is not resonant. This *præcordial dulness* does not correspond to the whole anterior surface of the heart, but to what is exposed between the vertical anterior edge of the right lung and the oblique anterior edge of the left; and not all of this, because the sternum is normally resonant even up to its left border. Its limits are as follows: Above, the upper border of the fourth costal cartilage; below, the upper border of the sixth cartilage; internally, the left border of the sternum; and externally, a vertical line from half an inch to one inch within the nipple. Around this dulness, above and to the right and to the left, is an area of less dulness, the outer limit of which corresponds to the outline of the heart, and therefore maps out its actual size; this reaches above the upper border of the third rib, or of the third space, to the left it reaches nearly the nipple line, and to the right the right border of the sternum; but the dulness is least marked over this bone. The former central area of dulness is often called *superficial*, or *absolute dulness*, the latter surrounding band is called *deep* or *relative dulness*. The former is best brought out by light percussion; the latter requires stronger percussion, and is sometimes better appreciated when the stethoscope is applied close by at the same time as the blow is struck (auscultatory percussion). The lower limit of the heart's dulness cannot be discriminated from that of the liver, and the outline is assumed to lie between the impulse and the lowest point of the right border of the dulness.

The deep or relative dulness varies with the position of the patient. In the erect position the upper limit is lower by a rib's breadth: and the transverse measurement is larger by more than three-quarters of an inch, the increase being greater at the right border. These differences, especially that in the transverse diameter, are much greater in some forms of cardiac disease, when the dulness may be from 2 to 4 inches wider in the erect than in the recumbent position (Gordon).

From the relation of the heart to the lung in this position, it results that the cardiac dulness is affected by changes in either of these organs. It is increased by enlargement of the heart, and diminished if the heart becomes smaller. It is, on the other hand, diminished by enlargements of the lung, which cover the heart; and it is enlarged by a retraction of the lungs, especially of the left, which exposes it more. An important cause of its enlargement is distension of the pericardial sac with liquid. Exceptionally, the area may be resonant from the presence of air in this sac. The

area of præcordial dulness is shifted upwards, downwards, or to either side by anything which displaces the heart in these directions.

AUSCULTATION

With the stethoscope we hear over the cardiac region, the well-known sounds of the heart; the *first*, or *systolic*, duller and longer, and the *second*, or *diastolic*, sharper and shorter. It is generally believed that the first is due partly to muscular contraction, and partly to closure of the auriculo-ventricular valves; and it is known that the second is due to closure of the sigmoid valves. The first sound is heard best near the apex of the heart, and the second is heard best at the base. In the erect posture, the sounds are more nearly like one another, the first being sharper, the second duller than it is in the recumbent position (Gordon).

Modifications of the Sounds.—The heart-sounds may be accentuated or diminished in loudness, or increased in number.

Accentuation arises from several causes, amongst others from retraction of the lung, so as to bring the heart closer to the chest-wall; and from increased tension in the aortic or pulmonary arterial system, whereby the valves are caused to close with unusual force. This last condition affects, of course, the second sound, and it may be determined whether the aortic or pulmonary system is at fault by examining successively on either side of the sternum in the second intercostal space. On the right side the aortic second sound can be heard more or less apart from the pulmonary; on the left side, the pulmonary apart from the aortic. Accentuation of the first sound results from excessive action of the heart, and is common also in mitral stenosis.

Diminution of the sounds results from feeble action of the heart, from its being unusually covered by lung, as in emphysema, or from its being surrounded by pericardial effusion.

When there appear to be *more than two sounds*, this is generally attributed to *reduplication* of the first sound, of the second sound, or of both; but it may be, as some believe, due to the introduction of other sounds, not belonging to the physiological group, or not usually audible. The best examples are the triple sound from apparent doubling of the first sound heard in conditions of high arterial tension, and especially in the hypertrophied heart of chronic renal disease (*bruit de galop* of French authors); and the triple sound with apparent doubling of the second sound heard in mitral constriction (*bruit de rappel*). Reduplication is commonly explained by want of synchronism in the closure or in the tension of the two auriculo-ventricular valves, or of the two sigmoid valves: but this explanation is not satisfactory; it seems to require asynchronism of ventricular action, a possibility not admitted by physiologists. Other explanations suggested are: dissociation of the valvular from the muscular element when the first sound appears doubled; modified conditions of vibration in mitral stenosis from the proximity

of the mitral to the aortic valves; an additional sound from aortic tension; and audibility of auricular contraction.

Murmurs.—These are adventitious sounds, which accompany or replace those which are physiological. Originally described as *bruits de souffle*, they are now commonly called *murmurs* or *bruits*, and they are due mainly to two causes. The first is, that any narrowing or *obstruction* of a cardiac orifice, such as is produced by vegetations on the valves, or by union of the valves together, will produce vibrations in the currents of blood forced through them. The second is, that if the valve does not perfectly close the orifice, some blood will flow back, or *regurgitate*, into the cavity whence it came; and this leaking of a small stream through a narrow orifice or chink will be accompanied by vibrations which, if of sufficient amplitude, are audible as sound. Such sounds are generally explained by the theory of the *veine fluide*. In either case, the blood passes through a constriction, or narrow orifice, into a wider space beyond; this determines the production of a jet, or "fluid vein," which breaks up in such a manner as to produce vibrations among its own particles. In the same way may be explained a murmur which is sometimes heard as the result of the passage of blood through a perforation in the septum between the ventricles. In some cases the conditions requisite for the production of a *veine fluide* do not seem to be fulfilled, and we must then suppose that eddies are produced which will cause sound-vibrations; in others, the sound may be due to vibration of the edges of the valve; in others, possibly, to impaction of a current of blood directly against an unyielding valve.

Murmurs differ from one another—(1) in time; (2) in their relation to the orifices of the heart; (3) in the character of the sound.

(1) Murmurs which are heard with the first sound, or between the first and the second sounds, occur during the contraction of the ventricles, and are called *systolic;* those which are heard with the second sound, or between it and the succeeding first sound, occur during the dilatation of the ventricle, and are called *diastolic*. Of these last, some begin at the very commencement of diastole and end before the next first sound; others commence a little later, but still end before the first sound; and others, again, begin after the second sound, and run up to, and finish, in the next first sound. These may be called respectively, *early, mid,* and *late* diastolic. The last is more commonly known as *presystolic*. In determining the rhythm of a particular murmur, its position should be noted in reference to the beat of the heart, or to the beat of the carotid artery, by the side of the thyroid cartilage. Either of these represents the systole of the ventricle with sufficient accuracy, but the radial pulse is a little later.

(2) It is sufficient to say here that murmurs of systolic and diastolic time may be heard, with varying frequency, in connection with all four orifices of the heart. The subject will be more fully discussed in the chapter on "Diseases of the Valves."

(3) The quality of the sound is most often blowing; it is sometimes rushing, sawing, or rasping. Sometimes murmurs have a distinctly *musical* quality. Half-detached fragments of valve playing in the blood-current, perforations in valves, and loose chordæ tendineæ sometimes cause such murmurs.

Variations of murmurs.—Murmurs vary with the position of the patient, probably from the effect of gravity upon the velocity of the blood-currents. Thus in the recumbent position there is an increase in the loudness of hæmic pulmonary systolic murmurs constantly, and often of aortic systolic, and mitral and tricuspid systolic murmurs; the hæmic murmur is sometimes heard only in the recumbent position. Conversely the erect position intensifies mitral obstructive murmurs, and pulmonary and aortic regurgitant murmurs.

In some cases a murmur, though not strictly musical, has a different *pitch* at one point from that which it has an inch away.

THE PULSE

The terms *pulse* and *pulsation* refer to such movements of alternating expansion and contraction as may be felt in any vessel of the body accessible to the finger, or in any structure or organ, such as the liver, sufficiently vascular to transmit these movements. These serve as an important means of ascertaining the action of the heart and the condition of the circulation. For observations upon the *arterial pulse* the radial artery at the wrist is commonly employed; but a pulse can also be felt in the ulnar artery at the wrist, in the brachial in the arm, in the carotid by the side of the thyroid cartilage, in the facial artery as it turns round the lower jaw, in the temporal artery above the ear, in the femoral artery below Poupart's ligament, in the posterior tibial behind the inner malleolus, and in the dorsalis pedis at the base of the metatarsal bones.

It should be remembered that the radial artery does not always lie in its normal situation, but sometimes turns over the radius to the back of the wrist, one or two inches above the joint; and this may happen on one or both sides. A minute communicating branch may sometimes be felt in its place; but, in any case, the absence of a pulse of the proper size may be very misleading unless the occasional abnormality be borne in mind. More rarely, the radial is abnormally small, and the comes nervi mediani compensates for this by its unusual size.

The features to be noted in the pulse are the *frequency* of the beats (*pulse rate*), the uniformity of their occurrence (*regularity*), the *volume* of the artery, the anatomical condition of the *arterial wall*, and the *arterial tension*.

Although these features can be recognised by the finger of the trained physician up to a certain point, minuter details can only be noted by means of instruments, of which different forms of sphygmograph and sphygmomanometer are in common use.

The Sphygmograph.—In this instrument a light spring presses upon the radial artery, and the movement of the artery wall is communicated to a lever; and this carries a fine point, which traces the magnified movements upon a blackened paper moving horizontally by clockwork. The pressure of the spring upon the artery required to give the true record varies with every case, and the best instruments register in ounces the amount of pressure employed.

Besides the frequency and regularity, which can be at once appreciated, there are other features of the record demanding special study. In the tracing of each beat of the arterial pulse (*see* Fig. 49) there is an upstroke, which is uninterrupted and almost vertical; and a downstroke, which is oblique, and interrupted by one or two elevations with intervening depressions.

The upstroke represents the contraction of the ventricles, driving blood into the aorta, and thereby causing a wave which is rapidly transmitted to the peripheral arteries. The apex of this upstroke has been known as the *percussion wave*. Its height is proportionate to the force of the ventricular contraction, and the quickness or suddenness of the contraction is indicated by the vertical course of the stroke. The height is also greater when the arterial wall is yielding, less when it is tense and resistant. Compare Figs. 49 A and 50 A, B, with Figs. 49 B and 51 A, B, C.*

Of the elevations in the course of the downstroke, the most constant is the *dicrotic wave* (Fig. 49 A, *c*, Fig. 50 A, *c*, Fig. 51 A, *c*). This is the same as can be felt with the finger in pulses that are called "dicrotic"; it is shown by the sphygmograph to be present in the majority of pulses, even when not perceptible to the finger. It is due to a reflected wave from the closed aortic valves and from the walls of the aorta. It is immediately preceded by a depression, the *aortic notch*, which corresponds to the end of the ventricular systole, and marks the closure of the aortic valves. The interval between the commencement of the percussion wave and the bottom of the aortic notch is thus the *systolic period* or *sphygmic period*. When the aortic notch reaches the base line (Fig. 50 A) the pulse is called *fully dicrotic;* it sometimes falls below the base line, and is then called *hyperdicrotic* (Fig. 50 B). In this case the percussion wave of the next beat appears to come before the dicrotic wave has completely passed, and indeed, the occurrence may be due to the increased rapidity of the beats. Dicrotism is best marked in soft pulses, with yielding and elastic walls; it is a common result of vasomotor paralysis as seen in highly febrile conditions (Fig. 50), and can be at once produced by the administration of amyl nitrite. It is diminished or abolished by conditions leading to hard pulse, such as Bright's disease, and by aortic regurgitation, in which case the reflection of a wave takes place imperfectly.

Between the percussion wave and the dicrotic wave—that is

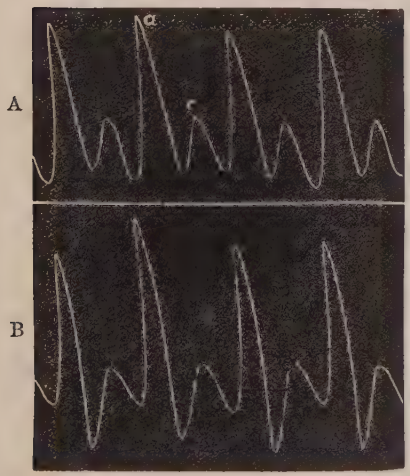
* The tracings were taken with a Marey's sphygmograph; a long and quick upstroke is curved backward, because the needle is at the end of a long lever, which works on a fulcrum, with an axis transverse to the line of movement of the paper.

preceding the aortic notch, and therefore corresponding to the period of systole of the ventricle—there is often a wave which has been attributed to the outward flow of the current of blood

FIG. 49

A. Normal Soft Pulse. Pressure Two Ounces.
B. Hard Pulse in Gout.

FIG. 50



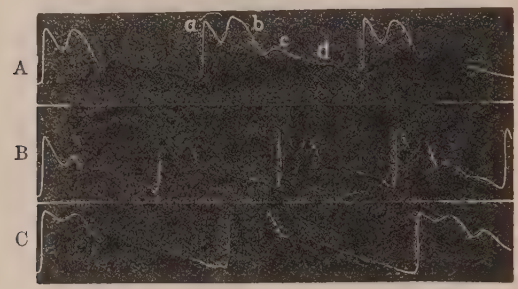
A. Dicrotic Pulse in Pyrexia. Temp. 102·2°.
B. Hyperdicrotic Pulse in Pyrexia (Enteric Fever). Temp. 103°.

following the percussion wave. It is called the *tidal* or *predicrotic wave* (Fig. 49 A, *b*, Fig. 51 A, *b*, B, C). It is best seen in hard pulses (Fig. 51)—that is, in conditions of high arterial tension, when it may be supposed that the undulations of the blood would be unusually well transmitted. On the other hand, in very soft pulses, the tidal wave is lost in the percussion wave (Fig. 50 A, B).

A pulse in which the tidal wave rises higher than the percussion wave has been called *anacrotic*, because the percussion wave forms an elevation in the ascending limb between the base and the highest point.

One or two slight undulations are occasionally seen after the

Fig. 51



A. Acute Bright's Disease. Pressure Four Ounces.
B. Acute Bright's Disease; Five Weeks' Duration. Pressure, Seven Ounces.
C. Chronic Bright's Disease. Pressure, Six Ounces.

dicrotic wave (Fig. 51 A, *d*). They occur in tracings of pulses of high tension only.

By some (Landois) the percussion wave and the dicrotic wave are regarded as the only primary waves, while the tidal wave and the undulations which follow the dicrotic wave are looked upon as secondary to these respectively, and are called *elastic elevations*. Others again (Von Frey and Krehl) say that all the undulations are reflected from the main (percussion) wave. These reflected waves are closer to the summit of the tracing, and more numerous, the higher the tension, which corresponds to the statement that high arterial tension is indicated by a high tidal wave, and by moderate dicrotism; whereas in low tension the dicrotism is considerable, and the tidal wave is small or absent.

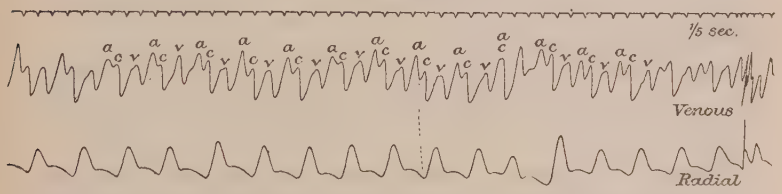
The resistance of the pulse to compression (*hardness* or *tension*) can be estimated by the amount of pressure required to be applied to the spring in order to get the most ample swing of the lever, such pressure varying from two to ten or more ounces; but for this estimate more reliance is now placed upon the different forms of sphygmomanometer. Special features are imparted to the tracing by some valvular diseases and changes in the walls of the vessels; the most characteristic are seen in aortic regurgitation, arterial obstruction, aneurysm, and atheroma.

When the heart's action is very feeble, the base line of the tracing may be undulating instead of straight. This shows the incapacity of the ventricle to overcome the influence of the respiratory movements upon the circulation, and is called the *respiratory wave*.

Venous Pulsation.—Some pulsation in the large veins of the body

appears to be a normal phenomenon ; and an undulating or distinctly pulsatile movement can be seen in the jugular veins, both external and internal, of some persons with quite healthy circulations ; but usually it is absent or inconspicuous. The relation of the movements to those of the radial pulse is often difficult to make out on

FIG. 52



A Polygraphic Curve showing the Three Waves of the Physiological Venous Pulse in the upper tracing, which was taken from the veins of the neck. The lower tracing is from the radial artery. With each systole of the ventricle are two waves, *c* and *v*. Preceding *c* in each cycle, and pre-systolic in time, a wave, *a*, is seen which is the result of auricular systole. (*T. Lewis.*)

simple inspection ; the external jugular may be seen just above the clavicle, and its movement differ from arterial movements in that the expansion is slow, while the collapse is more sudden, and corresponds nearly to the rise of the radial.

The internal jugular produces a large undulatory movement with slow rise and quicker fall over the side of the neck between the angle of the jaw and the sterno-mastoid muscle, and it must not be confounded with carotid pulsation.

More accurate information can be obtained by tracings from the jugular pulse by means of a tambour applied to it, the movements of which are transmitted to a needle writing either on a drum, or on the smoked paper of the sphygmograph, parallel with a radial tracing, as in the *polygraph* of Dr. Mackenzie. The tracing usually obtained shows three waves (positive waves), with, of course, intervening depressions (negative waves). The interpretation of these waves has been much discussed. The first positive wave (Fig. 52, *a*) occurs just before the sphygmic period of the radial tracing (*see* p. 568), and is admitted by all to be due to the right auricular contraction. The depression which follows it occurs during the sphygmic period, and is mainly due to the emptying of the blood into the relaxing and dilating right auricle. This fall is interrupted by the second positive wave (*c*), which is by some attributed to the projection of the auriculo-ventricular valve (tricuspid) into the right auricle during the contraction of the right ventricle ; by others to the carotid artery beating against the jugular vein. In either case it is recognised as representing a ventricular contraction ; and the interval between *a* and *c* is taken to be the measure of the time of conduction of the wave of muscular contraction from auricle to ventricle. In normal individuals, its duration is about

one-fifth of a second. The third wave (*v*) is either due to the filling of the jugular vein by passive flow when the auricle has become distended, or to an elevation of the auriculo-ventricular ring with the diastole of the ventricle.

In various conditions of disease the features of the venous pulse are altered : and by a comparison of the waves in the venous tracing with those in the radial tracing inferences can be drawn as to the condition of the cavities of the heart, in respect of the five functions above mentioned (*see* p. 562).

An important modification of the venous pulse is that which is seen in cases of regurgitation through the tricuspid valve ; and in these cases a similar pulsation can be often felt in the enlarged and congested liver.

Pulsation of a different kind is sometimes seen in the *peripheral veins,* especially those of the backs of the hands and feet, which is due to the transmission of the arterial wave through the capillaries to the veins. It results from great relaxation of the vascular walls, with a powerful or excited action of the heart; thus it may occur in febrile conditions, in the heat of summer, or after a full meal.

The Pulse-Rate.—In health the heart beats about seventy times in the minute, with variations between fifty and eighty.

Increased frequency.—Nearly every disturbance of health quickens the pulse ; and this increased rate is the constant accompaniment of acute and subacute inflammation, of fever in all forms, of states of exhaustion, and of most forms of cardiac disease.

Excessive slowness of the pulse occurs in some cardiac lesions, and especially aortic stenosis, or fatty heart ; in conditions of collapse, in meningitis and cerebral tumours, and in jaundice.

It is here necessary to point out that the radial pulse as felt by the finger may fail to record some ventricular contractions. If a ventricular contraction is very feeble, the consequent wave of blood may not be large enough to be felt by the finger ; but it may be recorded on a sphygmographic tracing. A still weaker contraction may scarcely lift the aortic valves, and so it may fail to impress even the sphygmograph. Moreover, the radial pulse has only to do with the left ventricle, and although in health every ventricular contraction is preceded by and is the immediate result of an auricular contraction, a comparison of jugular venous pulse tracings with radial tracings shows that in certain circumstances the ventricular contraction may fail every other auricular contraction ; or it may fail two beats out of every three ; or the ventricle may beat quite independently of the auricle—the condition already described as heart-block (*see* p. 562). There are also conditions (advanced mitral stenosis) in which, apparently, the auricle is paralysed as a contractile organ (auricular fibrillation) and the ventricle beats alone.

Irregularity.—There is an irregularity of volume as well as of time ; but the former is largely dependent upon the latter ; and with the latter we are mainly concerned here. In children there is often considerable variation in the diastolic periods of different

beats, producing a mild form of true time irregularity. This disappears in later life. In adults a common form of irregular pulse is what has been called the *intermittent pulse*. Here several successive beats come quite regularly, and then a beat is missed; and after an interval corresponding to the loss of this beat, the pulse occurs

FIG. 53

Diagram of Radial Pulse Tracing showing Premature Beat at x.

FIG. 54

Tracing from Radial Pulse with Intermission, the Premature Beat not reaching the Wrist.

again, and continues until another omission or *intermission* takes place. This happens every four or five beats; or more rarely, viz., every twenty, thirty, or forty; but in any given case the number of beats between the intermissions is not uniform. In the majority of cases, the sphygmograph shows that the heart does not really fail to beat, but that at this point the ventricular contraction occurs close to its predecessor, and that the wave in the radial pulse is too small to be felt by the finger; it has been called a *premature systole*, or *extra systole*. The diastolic interval after the premature systole is correspondingly lengthened, and is generally such that the beat before and the beat after are separated by a period of time equal to two ordinary intervals (*see* Figs. 53 and 54).

It is the length of this interval which gives the impression of a stoppage of the heart, or an intermission; but *extra systoles* not succeeded by long intervals, and sometimes following quickly upon one another, occur in many forms of heart disease both organic and functional (*see* Fig. 60, A, B). Every other beat may occur prematurely, so that it is closer to its predecessor than to its successor, and the beats occur in pairs (*coupled beats*, or less accurately, *pulsus bigeminus, twin-pulse*). A normal beat may be regularly followed by two extra systoles, when a triple beat will be observed (*pulsus trigeminus*).

Such an extra systole produces a wave of less amplitude than the normal wave preceding it; and the nearer it is to this preceding wave, the smaller it is. The explanation is that some abnormal stimulus to contraction of the ventricle arises in the "refractory phase," when the irritability of the ventricle is lowered, and so the stimulus has less effect than normal: the longer the interval after the normal wave, the greater the effect. After the extra systole

there is an abnormally long period due to the fact that the extra systole anticipating the next normal beat is followed by a refractory phase, either of the ventricle or of the auriculo-ventricular bundle, which prevents the response of the muscle to the next stimulus occurring in normal rhythm. The long repose of the ventricle enables its irritability to accumulate, so that the next normal stimulus produces an unusual contraction, as shown in the first rise after x in Fig. 53.

Extra systoles may be habitual, or temporary, or due to functional or toxic conditions, or to organic disease of the heart.

Extra systoles form an important element in the various forms of irregularity of the heart, whether functional or organic; and three varieties have been described, which can only be distinguished by a comparison of jugular with radial tracings. They are (α) extra systoles arising in the ventricle (auriculo-ventricular bundle below the node), which appear in the radial tracing, while the auricular contractions can be seen to be regular in rhythm; (β) extra systoles arising in the auricle: the early contraction of the auricles is shown in the jugular tracing; and (γ) extra systoles in which the auricle and ventricle appear to contract simultaneously and prematurely. Mackenzie suggests that in this form the stimulus arises in the auriculo-ventricular node; and he calls them *nodal extra systoles.* Usually the *ventricular* extra systole is followed by the long compensatory pause above described, whereas an auricular extra systole is followed by an interval no longer than the normal.

The *pulsus alternans* is one in which there is a regular alternation of large and small beats, but the intervals are almost exactly uniform, instead of varying alternately with the size of the wave, as in coupled beats or pulsus bigeminus. It is attributed to defective contractility.

The irregularity may be complete, *i.e.*, of such a kind that no order is observed at all: long, short, and shorter intervals follow each other indiscriminately, and the force and volume of the beats correspondingly vary. Such irregularity is seen commonly in mitral disease, and in dilatation of the heart, and is explained by the condition known as fibrillation of the auricle (*see* p. 599). An irregularity of less degree occurs in some cerebral lesions—*e.g.*, meningitis.

The Volume of the Pulse.—This is determined partly by the actual size of the artery, and partly by the quantity of blood sent into the artery at each beat of the heart. If much blood is sent in, the pulse is full or large; if little blood, the pulse is small.

In an irregular pulse the beats generally vary in volume as well as in the time of occurrence; for a long diastolic period gives time for the muscular contractility to accumulate, and hence the next contraction is often more forcible (*see* Figs. 53, 54, 60, A, B).

In the *pulsus paradoxus* there is a considerable diminution or complete absence of the pulse during inspiration. Though rare, it may occur under several conditions, such as mediastino-pericarditis,

pericarditis, mediastinal tumour, great cardiac weakness, pleural effusion, or obstruction of the air-passages.

The Arterial Wall.—If the pulse be stopped by the pressure of the finger, and emptied of blood, it should in health be scarcely perceptible as a separate structure; if it is thickened or rigid from arterio-sclerosis it is easily felt; and if highly calcareous, the irregularities of the deposit can be felt on passing the finger up and down its length.

Hardness of the Pulse: Arterial Tension.—If the finger be pressed upon the artery with increasing force, the flow of blood is at length stopped; and this arrest of the blood-flow can be more easily effected in some pulses than in others, apart from changes in the arterial wall. Those in which slight pressure is sufficient are called *soft* or *compressible* pulses; those in which much pressure is required are called *hard* or *incompressible* pulses. If when the pulse has been stopped by compression the finger be slowly lifted, the blood will be felt to pass under the finger with much greater force in the case of the hard than in the case of the soft pulse. A more certain estimate of the hardness of the pulse can be derived from the use of the sphygmograph above described, and from different forms of sphygmomanometer.

The Sphygmomanometer.—In this instrument, of which there are several varieties (Riva-Rocci, Erlanger, Gibson, Hill and Barnard), the arterial pressure is measured by its displacing effect upon a column of mercury. In most instruments the upper arm is encircled by a broad double band or bag of india-rubber into which air can be forced by an india-rubber ball and valve through a connecting rubber tube; another tube proceeding from this is connected with the manometer, and the pressure in the india-rubber bag is measured in millimetres by the difference in height of the two columns of mercury. When the instrument is adjusted, air is pumped into the armlet and manometer until the pressure is more than sufficient to stop the pulse at the wrist. The air is then gradually allowed to escape, until the pressure is reduced to a point which just allows the pulse to be felt. The figure in the scale at which the mercury then stands represents the *systolic pressure*. The reading of *diastolic pressure* is held to be given at the time when, air still escaping, the pulse is for the first time felt beating freely at the wrist. The difference between the systolic and diastolic pressures is from 25 to 30 mm.

If a float carrying a needle be placed on the surface of the mercurial column, the movements of the column can be recorded on the drum of a kymographion; and by the use of a tambour placed on the radial artery, or on the brachial artery below the armlet, and connected by a rubber tube with another lever, the exact point at which the blood current is stopped, or recommences after stoppage, can be recorded on the same paper. This is effected in the instrument of Dr. G. A. Gibson, and in a similar more convenient one designed by Dr. C. Singer. As the mercury

falls after the escape of the air from the armlet an oblique undulating line is traced on the revolving cylinder. When the blood forces its way into the vessel below the armlet, as recorded by the second needle, the height of the mercurial column in millimetres (doubled because it is moving in a U-shaped tube) represents the maximum or systolic pressure. The minimum or diastolic pressure is represented by the level at which the greatest amplitude of pulsation is recorded. In some apparatus a recording dial is substituted for the manometer tube, and an instrument, known as an *oscillometer,* in which the principles of the aneroid are utilised, records both systolic and diastolic pressures.

In healthy persons the systolic pressure increases from childhood to old age. From 8 to 14 years, it is equal to 90 mm. of mercury; from 15 to 21 years, 100 to 120 mm.; from 21 to 65 years, 120 to 135 or 150 mm.; and above 65 years, 135 to 150 mm. In women it is from 10 to 15 mm. less than in men of the same age (Brunton). In arterio-sclerosis and in Bright's disease it may be 180 or 200 mm. or more. There is much difference of opinion as to whether, when the artery wall is thickened, a high record is partly due to such thickening, or is solely due to the blood-pressure.

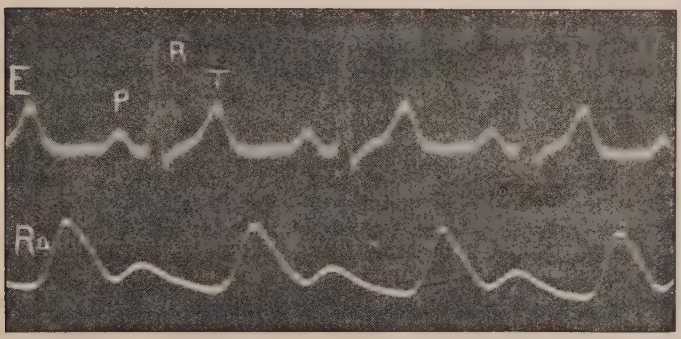
A hard pulse is not necessarily very small or very large. It feels like a cord, even though there are no changes in the artery wall, but, of course, the two conditions, rigidity of arterial wall and hardness of pulse, may co-exist. A pulse is hard in proportion to (1) the quantity of blood thrown into the arterial system, (2) the difficulty of egress through the arterioles, capillaries, and veins (often spoken of as capillary resistance), and (3) the degree of contraction of the arterial coats upon their contents. It is soft under the opposite conditions. Thus, hardness is favoured by a powerfully acting heart, a normal amount of blood, contraction of the peripheral arterioles—as, for instance, by cold, which stimulates the muscular coat of the arteries (vasomotor stimulation). Softness of pulse is favoured by a feeble heart, by valvular imperfections interfering with the supply of the blood to the arterial system, by a free flow through the capillary area, and by dilatation of the arteries and arterioles as a result of vasomotor paralysis. The hard pulse is indicative of *high arterial tension;* the soft pulse of *low arterial tension.* The former is often accompanied by an accentuated aortic second sound, heard at the inner end of the second right intercostal space, and sometimes by a reduplication of the first sound, heard over the septum ventriculorum.

Dicrotism.—This feature of the normal pulse (*see* p. 568) can only be appreciated by the finger when it is well marked, and that mostly in febrile conditions; the dicrotic wave may then be so large as to make one beat seem like two. A careful comparison with the heart by palpation and auscultation will prevent a mistake.

EXAMINATION OF THE HEART BY THE CARDIOGRAPH

Cardiographs have long been in use, constructed on the same principle as the sphygmograph; that is, the movements of the apex, or other part of the cardiac surface lying close to the thorax, are recorded by a spring, or by an air-tambour, conveying the movements to a needle, which records them upon a drum or paper slip moved by clockwork. On account of the fact that the auricles, ventricles, and large vessels are all in movement together, and that the record at any one point of the surface of the heart is affected

FIG. 55



An Electro-cardiogram, taken from a Patient with the String-galvanometer; and a Radial Curve. Each heart cycle is accompanied by three chief electric variations, P, R, and T respectively. P is the result of auricular, R and T are the result of ventricular contraction. Ra is the radial curve.

by the movement of remoter parts, the interpretation of the results has always been difficult, and the use of the instrument has accordingly been very limited.

More definite information has been obtained by the polygraph, by which the auricular and ventricular events can be better differentiated; and a similar purpose is served by the method now to be mentioned.

Electro-cardiograms.—These are obtained by means of the string-galvanometer, an instrument in which an exceedingly fine platinum wire is fixed vertically between the poles of a powerful electro-magnet. A microscope is brought to bear upon it, and by a special arrangement its slightest movement can be recorded by a photographic apparatus. The poles of the magnet are placed in water, in which at the same time preferably one arm and one leg, or the two arms of the patient are placed. Electric currents due to the contraction of the heart's cavities are transmitted to the magnet, and cause a lateral movement of the fine vertical wire.

These movements are represented as deviations from the base line on a strip of photographic paper. The best results from the auricles appear to be given by the upper limbs, and the best from the ventricles by the lower limbs. Fig. 55 is a tracing kindly supplied by Dr. Thomas Lewis.

AUSCULTATION OF THE ARTERIES

If the carotid or subclavian artery is auscultated without pressure by the stethoscope two sounds are usually heard—a systolic sound due to expansion of the vessel, and a diastolic sound which is the conducted aortic second sound. The first of these is sometimes absent. Over the abdominal aorta and the femoral artery a systolic sound is heard like the above. In the other arteries, as a rule, nothing is heard. If the stethoscope be pressed upon a large artery, a systolic *pressure murmur* is heard; if the pressure is increased so as nearly to close the artery, the murmur is changed into a *pressure sound;* if the artery is quite closed there is silence.

AUSCULTATION OF THE VEINS

If, in very anæmic persons, and in healthy children, the stethoscope be placed over the lower part of the jugular vein, at the point of separation of the sternal from the clavicular attachments of the sterno-mastoid, a continuous humming or rushing noise will be heard, which has been called the venous hum, or *bruit de diable,* from a French toy, called "diable," which makes a similar noise. This murmur is heard best in the erect posture, with the head turned away from the side which is being examined. This could be explained on the theory of the *veine fluide,* if the quantity of blood were less than normal, by supposing that the upper part of the vein adapts itself to the lessened amount of blood passing through it, while the lowest part, or ampulla, is kept in its natural state of dilatation by cervical fascia; blood would then pass from a narrow space to a wider space beyond. But in chlorosis at any rate the blood is not less in quantity, nor, as far as I know, is it in healthy children, in whom, nevertheless, a *bruit de diable* is very frequent. In children, too, the venous murmur is sometimes heard over the manubrium sterni, and this especially if the head be thrown back with the face looking upwards.

DISEASES OF THE HEART

ENDOCARDITIS

In considering the diseases of the heart, it is convenient to deal separately with the *endocardium*, the muscular substance, or *myocardium*, and the *pericardium*; and the first place must be given to the endocardium, on account of the important share which its inflammatory lesions, involving as they do the valvular apparatus, have upon the mechanism of the heart, as the organ of the circulation.

Endocarditis, like so many other inflammatory processes, is probably always due to the action of micro-organisms or their toxins. As a rule the parts first affected are the valves of the left side of the heart: the lesion is often confined to them, and may completely subside, or if any traces are left they consist of structural damage to the valve of which the later consequences are solely dependent on the mechanical failure of the valve. This is a simple *acute endocarditis*. In other cases, more extensive changes take place in the valves, micro-organisms are present in great numbers, and being conveyed by the current of blood to remote parts of the body, set up fresh foci of disease—*malignant endocarditis*. The term *chronic endocarditis* is applied to the permanent deformities and changes in the valves which result from a simple acute endocarditis, as well as to a separate inflammatory process of slow development.

ACUTE ENDOCARDITIS

Ætiology.—Endocarditis is, in the great majority of cases, a part of rheumatic fever (*see* p. 168); it also occurs during the progress of chorea, and in scarlet fever, diphtheria, typhoid, and some other infectious diseases. Bright's disease, syphilis, and other chronic dyscrasiæ, are said occasionally to cause it. It may be brought on by local injuries, such as the rupture of a sigmoid valve, or of the chordæ tendineæ, and the unnatural friction of one part of the heart with another; and the passage of currents of blood through abnormal apertures may cause the local inflammation of the endocardium.

It is always in limited patches, and never affects the whole interior of the heart. In rheumatic fever and other general diseases it affects the valves first or alone.

The relation of endocarditis to the two sides of the heart is of very great importance. If endocarditis occurs during fœtal life, it is believed to attack the pulmonary or tricuspid valves; but, with this exception, simple acute endocarditis is almost invariably on the *left* side. Hence what follows in this chapter chiefly concerns the aortic and mitral valves.

Anatomical Changes.—The earliest change is a very slight swelling of the subendocardial tissue near the edge of the valve, on the auricular side of a mitral cusp, or the ventricular side of an aortic cusp, so as to form a number of beadlike elevations, usually described as *vegetations;* and these occupy at first the line between the portion of the valve which touches its fellow on closure, and the portion which is free. The swelling results from œdema and infiltration with lymphocytes, the consequence in rheumatism, according to Carey Coombs, of micro-organisms brought into the base of the valve by the coronary arteries. He also finds in the inflamed valves nodules containing large multinuclear cells. Later the endothelium of the valves over the inflamed area becomes loosened, and fibrin and polymorphonuclear leucocytes are deposited from the ventricular blood. By the continued addition of deposits of fibrin, very large vegetations may be formed which project into the valvular orifice, and from which particles, loosened by softening, may be detached by the force of the current of blood. Such a detached particle is carried along with the blood-current to distant vessels of gradually diminishing size, and ultimately meeting with one small enough to resist its further progress, becomes impacted therein. This process, known as *embolism*, is an important element in malignant endocarditis. But at different stages short of this, subsidence of the inflammation may occur, and in the earliest period complete resolution probably may take place; there is, however, more often some formation of vessels, organisation of the infiltration, and growth of fibrous tissue, by the gradual and irregular contraction of which—a process similar to that which occurs after inflammation in other parts—the valves become shortened, deformed, and incapable of completely covering the orifice they are intended to close. In some cases the fibrous tissue acquires an almost cartilaginous hardness, or calcareous particles are deposited, and the valve segments not only fail to close the valve aperture, but, by their constant projection into the orifice, offer a definite obstruction to the passage of the blood through it.

In acute stages, streptococci, staphylococci, pneumococci, and diplococci have been found; but they are absent from the chronic lesions.

Symptoms.—Endocarditis, as it occurs in rheumatic fever, has but few symptoms. Indeed, it mostly proceeds without any appreciable alteration of those which are due to the rheumatism, and is detected only by the stethoscope. It happens in somewhat less than half of all cases of rheumatic fever, and mostly within seven days of the beginning of the attack. If the heart be carefully auscultated, the first indication will be a slight prolongation, or roughness, or some want of clearness of the first sound in the aortic or mitral area, according as the one or the other valve is the seat of inflammation. Within twenty-four hours it may lengthen into a distinct murmur, or soft blowing sound, which accompanies, and does not abolish, the first sound. If the aortic valve is affected, the

second sound may become imperfect, and a diastolic murmur may become developed, but this is much less frequent, while the systolic murmur in the mitral area is the most common of all. As above stated, this physical sign may be the only indication of endocarditis, but increased force and frequency of the heart's action, palpitation, and some præcordial pain and distress are occasionally observed. The evidences of endocarditis persist for a variable time; the murmur may disappear entirely in the course of the rheumatic attack; or it may become louder and harsher, more widely diffused, or definitely follow the course of the blood-current, showing the existence of valvular obstruction or incompetence.

Diagnosis.—This requires some care, as the murmurs of recent acute endocarditis may be confounded with *functional* murmurs, with the murmurs of old *valvular disease*, and with *pericardial* friction sounds. The chief point to note is that the murmur of acute endocarditis is generally soft in quality, systolic in time, and strictly limited to the area of the valve affected—that is, either the aortic or the mitral area. Acute simple endocarditis of the pulmonary or of the tricuspid valve is practically out of the question. A functional or hæmic murmur is generally loudest over the pulmonary artery, and often harsh in quality. The murmur of chronic valvular disease is often loud or harsh, heard over a large area, and accompanied by some alteration in the size or shape of the heart.

Prognosis.—In the course of an attack of rheumatic fever, or of any other illness causing acute endocarditis, there is nothing to guide one as to the outcome of the disease. In a large number of cases the murmur disappears, and the patient apparently recovers completely; in a certain proportion of these, nevertheless, valvular disease supervenes several years afterwards. In a few cases the affected valve soon becomes incompetent, and the patient suffers henceforth from "heart disease."

Treatment.—The influence of treatment upon acute simple endocarditis is not very apparent, and many are content to leave it alone. Sometimes a small blister, about three inches by two inches, is placed on the chest over the affected valve. The treatment proper to acute rheumatism should be continued, and this will, of course, necessitate rest in the recumbent position and light diet, which are also desirable for endocarditis.

MALIGNANT ENDOCARDITIS

(*Septic, Infective, or Ulcerative Endocarditis*)

Ætiology.—Acute rheumatism is an antecedent of malignant endocarditis, but the proportion of cases (53 out of 160—Osler) is less than that in which rheumatism is related to simple endocarditis: in some of these the symptoms have developed in the course of the rheumatic fever, and in others they have arisen in

the stage of chronic valvular disease, which has an important influence in the occurrence of infection. Malignant endocarditis may occur quite spontaneously—at any rate, without any previous history to explain it in the present state of our knowledge. On the other hand, besides rheumatism, its predisposing cause may be found in acute pneumonia, in the eruptive fevers such as scarlatina, in puerperal processes, in the existence of open wounds on the surface of the body, in purulent discharges from the mucous membranes (urethritis, vaginitis), in septicæmia and pyæmia, in malaria, and in some other conditions. Various micro-organisms are found in the organs in infective endocarditis. Streptococci, staphylococci, and pneumococci are most common: the bacillus pneumoniæ of Friedländer, and the bacilli of tubercle, diphtheria, and typhoid have also been found, as well as some organisms which are not present in other diseases. From some centre of infection, the organisms find an entrance into the blood and are thence deposited on the valves. Streptococci are sometimes found in the blood during life.

Anatomical Changes.—In this form of endocarditis the tissue of the inflamed valve is softened, and breaks down, so that erosions or ulcerations take place, and, as a result of this, fibrin is deposited upon the roughened surface, and accumulates into irregular masses of vegetations, which may reach the size of a hazel-nut. By suitable methods the micro-organisms can be demonstrated on the surface, and more or less deeply in the substance of the vegetations and fibrinous deposits, where they form considerable masses or colonies. Several important changes result from these processes in the valve. The valve itself may be perforated, or strips of tissue may be partly separated and hang loosely in the blood-current, or portions may be completely detached. Sometimes a part of the valve is so weakened by the destructive process that it yields before the pressure of the blood, and a saccular dilatation, or *aneurysm*, of the valve is formed, projecting on the opposite side. Another result is the occurrence of endocarditis, or endarteritis, in adjacent parts from a strip of the valve playing backwards and forwards in the blood-currents with the systole and diastole of the ventricle, and striking alternately the walls of the cavities in front and behind. In the case of mitral endocarditis, these are the left ventricle and the left auricle; in the case of aortic endocarditis, they are the aorta and the left ventricle. At the spot struck infection takes place, and causes a fresh patch of inflammation of the lining membrane.

But the most important effect of malignant endocarditis is the infection of the whole arterial system by particles detached from the valves being carried to remote parts; and it is to this process, combined with the presence of organisms in the detached fragments, that the special features of this disease are due. Embolism may take place in almost any part of the body. It is especially common in the vessels of the spleen and kidneys, but it happens

also in the vessels of the brain, alimentary canal, skin, retina, and lungs, and the larger arteries supplying the limbs, such as the radial, ulnar, tibial, brachial, and others. The local results of these impactions are: (1) Obstruction of the circulation; (2) necrosis or hæmorrhage, or both, within the area of distribution of the obstructed vessel, and the formation of infarcts; and (3) suppuration in the same area from the septic influence of the micro-organisms (*see* Embolism).

The effects upon the various organs, as they may be seen in different cases of malignant endocarditis, are—softening and abscess of the brain, and meningitis; retinal hæmorrhages and optic neuritis; diffused swelling, infarction, and abscess of the spleen; infarction or general diffused inflammation of the kidneys, which are often large, and finely mottled or speckled with hæmorrhagic points upon a white ground; hæmorrhages under the skin; hæmorrhagic infarctions and abscesses of the lungs; pleurisy and empyema.

Malignant endocarditis, like simple endocarditis, affects chiefly the left side of the heart; but the proportion of cases in which the right side is involved is much larger than in the simple form.

Symptoms.—The symptoms and course of the disease present the greatest variety. In some cases the disease is at first simply the occurrence of fever with afternoon rises of temperature, or perhaps sweating, in a patient living an active life, though perhaps known to have valvular disease, more or less perfectly compensated. The temperature may be high, reaching 102° or 103°; but it is generally remittent or intermittent, and sometimes with remarkable regularity for long periods. There is often free perspiration, and there may be an occasional rigor. The pulse is rapid, ranging from 90 to 120, or even higher. If the heart be auscultated, a murmur will generally be heard at one or other orifice, mostly, however, on the left side. Still, it must not be forgotten that in these cases murmurs may be entirely absent. In the cases with a previous history of rheumatism, or known cardiac disease, there may be abnormalities in the size and action of the heart. The respirations are rapid, sometimes without definite lesions of the lungs, at others with signs of bronchitis, œdema, or congestion.

Such cases may go on for several months (*chronic infective endocarditis*) with little variation and little deterioration, but at length grow weaker and die, or suffer from one or more of the several complications mentioned below.

In a considerable number of cases there is a close resemblance to *typhoid fever*, chiefly on account of the almost spontaneous occurrence of fever, with headache and perhaps enlarged spleen.

Thus, the patient may have been perfectly well until he complains of some such symptoms as usher in other severe febrile diseases, pain in the head, or in the back and limbs, or a definite rigor or rigors. Then follows severe pyrexia, with its usual conditions—high temperature, quick pulse and respiration, dry tongue, loss of appetite,

thirst, and malaise. Frequently, within a few days, the patient is prostrate, apathetic, drowsy, and at night delirious; but the time of appearance of this symptom, determined presumably by the virulence of the toxin, is variable.

The condition of the bowels varies, but there are often loose yellow motions, with much resemblance to those of typhoid fever; and the abdomen may be distended.

The spleen is enlarged either from the general infection or from embolism; but there are no rose-spots. The duration in these cases is generally much less than in cases of chronic infective endocarditis, namely, from ten days to two or three weeks.

In another group of cases rigors are a prominent symptom, occurring once, twice, or more times in the day, and the resemblance to *pyæmia* from wounds is very close. Rheumatism and actual cardiac disease are less often present as antecedents, and the endocarditis more often affects the right side of the heart than in the typhoid form. It may begin, like the last, with vague or more decided pains in the limbs or back, until the first rigor occurs; and intense anæmia is often present. The heart may present but little or no evidence of enlargement, and the murmur may be limited to the area of the pulmonary artery, in which case it may be thought that the murmur is simply hæmic, as a consequence of the pronounced anæmia; on the other hand, in many cases the same conditions of the cardiac apparatus may exist as in the other forms —namely, the murmurs of mitral or of aortic disease. For the rest, the local conditions are not distinctive. The spleen may be enlarged, diarrhœa with loose yellow motions often occurs, and albuminuria may be present. Sometimes the joints inflame or suppurate. The temperature rises to a great height, 105° or 106°, in the rigors, and it may fall to the normal or subnormal in the intervals. Sweating occurs, and is often profuse. As the case continues, emaciation becomes more marked, and with increasing delirium and apathy or coma, death results. These cases are often of short duration.

In again another group the organisms invade the cerebral meninges, and the symptoms of meningitis form the prominent feature. This often occurs in association with pneumonia, and the case is a combined pneumococcal meningitis and pneumococcal endocarditis. The cerebral symptoms consist of headache, convulsions, and coma, and death may take place quickly after their onset. The meningitis is often basal, and the exudation consists of greenish lymph or pus, containing pneumococci.

Marked cerebral symptoms, with a finely petechial eruption, may cause the case for a time to resemble typhus.

The symptoms of a general infection with micro-organisms from the cardiac valves may arise at any period in the history of a case of cardiac valvular disease. In chronic sufferers, confined to bed by dropsy or other results of cardiac failure, the change to a malignant type of endocarditis is indicated by remittent pyrexia,

and by the occurrence of embolism in different parts of the body; but the cardiac symptoms may continue predominant, and thus this group of cases may be distinguished from those first described.

In any case of the disease, to the symptoms dependent upon septicæmic infection may be added those due to the obstruction by embolism of arteries or arterioles.

Sometimes embolism of a large vessel in the brain will occur, and cause hemiplegia; if a vessel in the leg or arm is obstructed, there will be loss of the pulse at the wrist or ankle; but neither gangrene nor coldness need occur, unless a very large vessel is involved. More frequent are embolisms of the small vessels in the viscera; there is thus often enlargement and tenderness of the spleen due in part to infarcts, and the spleen may weigh from 20 to 30 ounces. Infarcts also occur in the kidney, accompanied, it may be, with pain, and the appearance of albumin or blood in the urine. In some cases petechial hæmorrhages appear under the skin, the petechiæ being generally small, and situate on the trunk, about the groins and axillæ; exceptionally a *purpuric* condition may be present for months. Hæmorrhages are often seen in the retina, and there may be also hæmoptysis or epistaxis. Some inflammatory conditions are probably also referable to embolic processes, such as the form of nephritis already mentioned (*see* p. 583), though whether as the result of vascular obstruction or of the introduction of micro-organisms may be doubtful; albuminuria from nephritis or infarct is frequent. Another important sign in cases of some length is pronounced anæmia, which occurs even when there has been no hæmorrhage. Optic neuritis is sometimes present. The tongue shows the usual changes of febrile diseases, being at first moist, with white fur, subsequently dry, glazed, or brown and cracked. In the more advanced stages, low delirium is mostly present, at first by night only, later on continuously; and this may lapse into complete coma before death.

The duration of malignant endocarditis is very variable. Some cases last six or seven months with little else than a constant pyrexia: cases of a pyæmic or severe typhoid type, and those with meningitis, are often fatal in a few weeks or days.

Diagnosis.—This rests upon the pyrexia of remittent or septic type, the existence of valvular disease, and the evidences of embolism above enumerated; marked anæmia and optic neuritis, if present, are also valuable signs. The heart should be examined in all cases of pyrexia of uncertain origin. But a murmur may be wanting throughout the whole illness; and even if present the valvular disease does not exclude the possibility of influenza, typhoid, or tuberculosis. Thus the diagnosis may often have to depend upon the occurrence of embolisms. Sometimes streptococci can be found in the blood. Malignant endocarditis is frequently mistaken for *typhoid fever*. Both are acute severe febrile diseases, and in both there may be a marked absence of localising symptoms; on the other hand, there is often in malignant endocarditis swelling of

the abdomen, with frequent loose yellow motions; and enlargement of the spleen may further increase the resemblance. The following are points of difference: In malignant endocarditis there is a more uniformly remittent or intermittent course of temperature, which may last several weeks; rigors, petechiæ under the skin, much pallor of the face, optic neuritis, or retinal hæmorrhages, if present, are in favour of endocarditis.

In some of the chronic cases, the spleen may be so enlarged, and the anæmia so considerable, that *splenic anæmia* is simulated: this is all the more likely if there are petechiæ or hæmorrhages from the mucous membranes and if the murmur is not unmistakably organic.

In the present day the occurrence of rigors should make one think as readily of malignant endocarditis as of the relatively infrequent *pyæmia*. In the latter, as a rule, the patient has more frequent rigors, an earthy colour of the face, and the evidences of inflammation at the base of the lung. But a wound may be the cause of septic endocarditis; and the diagnosis is further complicated by the fact that the endocarditis may itself be only a part of a typical pyæmia. *Malaria* may also be suggested by septic endocarditis: its presence would have to be confirmed by paludal associations, by the presence of plasmodia in the blood, and by the effects of quinine. A continued pyrexia without obvious signs may be due to commencing *miliary tuberculosis* as well as malignant endocarditis; but after a time local signs peculiar to one or the other ought to be observed.

One should always be alive to the possibility of septic conditions arising in the course of old heart disease, whether the patient be active or bedridden. Marked anæmia, continued pyrexia, rigors, prostration out of proportion to the valvular lesion, and the evidences of embolism in kidneys, spleen, or brain, are the facts that should guide the physician.

Prognosis.—This is exceedingly bad, and the recovery of a well-marked case of either typhoid or pyæmic form is rare. On the other hand, attacks of pyrexia in old valvular disease, with or without evidence of embolic processes, have subsided sometimes, to occur again after an interval of weeks or months.

Treatment can obviously be little more than palliative in the majority of cases. As in pyæmia, if there is any wound or sore, it should be rendered aseptic, and an attempt may be made to influence the disease by frequent doses of quinine (5 gr.), sodium sulphocarbolate (10 to 20 gr.), or sodium benzoate (20 gr.). In a few cases good results have followed the subcutaneous injection of an antistreptococcus serum, or of a *vaccine* prepared from the organisms actually found in the blood, of which organisms from fifty millions to one or two hundred millions are injected. But they have more frequently been found to be useless. The general rules for nursing and dieting in typhoid fever are applicable here: milk, beef-tea, and other light nutriment being given frequently in small quantities. Profuse diarrhœa may be checked, if required,

by astringents. The delirium is rarely so violent as to require any special treatment. Stimulants are naturally given, as the heart's action early tends to be seriously affected.

CHRONIC ENDOCARDITIS AND DISEASES OF THE VALVES

Little is known *clinically* of the course of chronic inflammation of the cardiac valves. What is called chronic inflammation is the thickening, shrinking, or deformity of the valve which is found after death, and which has been often preceded years before by rheumatic fever with accompanying evidence of acute endocarditis, or by rheumatic fever without such evidence. In the latter case it is generally assumed that a slow inflammatory change has taken place, giving rise to no symptoms until a certain degree of structural, generally fibrous, change has occurred.

As seen in the aortic valves, the changes consist in thickening of the base of the cusps, and to a less extent of the free edge, with shortening of the radial measurements, so that it is obvious that the cusps cannot meet to cover the orifice. The valves may be so fused together and thickened as both to present an obstruction to the flow of blood, and to prevent complete closure. Exceptionally the fusion is so complete as to allow only a small opening for the passage of blood into the aorta: and yet the valve may close well; but this is very rare.

The mitral valve is liable to similar changes: in some instances, some thickening and shortening of the cusps, so that complete adaptation is not produced; in many more cases the two cusps are fused together and much thickened, while the chordæ tendineæ are thickened and shortened—so shortened, indeed, that the united cusps are continuous with the musculi papillares, and these themselves are invaded by the fibrotic change. In some cases the cusps are united at the free edges, leaving the major part thickened and rigid in varying degree: in other cases the cusps are so closely fused together, that only a narrow slit presents itself on the auricular side in the dense surface of the valve. A distinction has thus been drawn between *funnel-shaped* and *buttonhole* orifices; and the former appears to be very much more frequent in children (8 to 1, Allbutt), the latter in adults (25 to 1).

The pulmonary valve sometimes presents changes suggestive of chronic endocarditis, rarely as a result of acquired disease, nearly always as a congenital lesion. It is very doubtful whether this is a truly inflammatory lesion (*see* Congenital Malformations). In the tricuspid valve lesions of the same nature as those of the mitral valve are seen, but they are much less extensive as a rule; only rarely causing an obstruction like that which is so common on the left side of the heart.

It will be understood that as a result of the above structural

changes, the efficiency of the valves must be seriously impaired, and the circulation of the blood through the heart must be to a greater or less extent affected. This takes place in two ways: first, the thickening of the valve, or vegetations or fibrinous masses upon it, or the union of two or more segments of a valve together, materially narrows or constricts the orifice through which the blood passes from auricle to ventricle, or from ventricle to aorta. Secondly, the contractions or deformities of the valve so shorten them or diminish their area, as to render them incapable of closing the orifice, and preventing reflux of blood from ventricle to auricle, or from aorta to ventricle. The one case is called *obstruction* or *stenosis*, the other *regurgitation* or *incompetence*. These two conditions may occur singly or combined at any one of the four orifices of the heart; but they are much more frequent on the left side of the heart than the right, because endocarditis, which is the chief cause of the valvular deformities, rarely attacks the right heart.

In connection with the effect of lesions attributable to chronic endocarditis upon the circulation, it is desirable to point out that changes in the valves and neighbouring structures arising from causes other than inflammation, may equally interfere with the proper action of the valves, and equally disturb the circulation and lead to all the secondary lesions which will in due course be described. Thus at the aortic orifice ruptures of the segments of the valves from strain and injury will lead forthwith to regurgitation, while secondary endocarditis and deposits of fibrin upon the injured valve will cause obstruction in addition. Atheroma, arteriosclerosis, and syphilitic endarteritis are fertile causes of lesions not only in the aortic valves, but in the aortic wall adjacent, by which the functions of the valves are prejudiced, chiefly in the direction of incompetence. These operate to a less degree in the case of the mitral valve. Another very frequent cause of regurgitation through an orifice is dilatation of the valvular ring or orifice from loss of tone in the muscles of the adjacent cardiac cavity, while the valve-cusps themselves are healthy; and this loss of tone follows upon abnormally high tension of the blood in the cavity.

Thus increased tension in the left ventricle as a result of aortic valve disease, causes yielding of the mitral orifice, and consequent regurgitation: high tension in the right ventricle resulting from mitral stenosis, or from chronic pulmonary disease, causes yielding of the tricuspid valve, and hence regurgitation. On the other hand, obstruction of an orifice is, very rarely, the result of a tumour projecting into an orifice; or of a mass of thrombus.

Relative Frequency of Valvular Lesions.—Mitral valve disease is more common than aortic valve disease. At the mitral orifice, regurgitation alone is the most frequent, a combination of obstruction and regurgitation next in frequency, and pure obstruction least frequent. However, the majority of cases of mitral disease of whatever kind coming to autopsy are found to have stenosis. At the aortic orifice, double disease (obstruction and

regurgitation) is most common, simple regurgitation comes next, and pure obstruction is comparatively rare. Mitral regurgitation is often the result of, and then accompanies, aortic disease. On the right side of the heart, tricuspid regurgitation is the only form that is at all frequent, and it is mostly secondary to mitral disease, or to chronic lung disease, such as emphysema or bronchiectasis. It may also follow mitral disease, when this is the result of aortic disease.

Effects upon the Heart.—The ordinary phenomena and symptoms of valvular disease of the heart are the effects, direct and remote, of the obstruction and regurgitation which accompany them. Valvular disease may occasionally exist for even long periods without producing any apparent result, except the physical sign of cardiac murmur, by which its presence is detected. The reserve power in the heart is sufficient to overcome the slight obstruction which the valve disease has created. But sooner or later the obstruction at the orifice, or the imperfect action of the valve, leads to a structural change in the walls of the heart. In accordance with what takes place in other parts of the body, the increased work thrown upon the walls of the left ventricle, in order to overcome an obstruction existing at the orifice of the aorta, leads to its *hypertrophy*, so long as it is adequately nourished, and up to a certain limit, which, no doubt, varies under different circumstances. If the resistance in front be carried beyond this limit, or if the muscle of the heart be insufficiently nourished, then the walls of the cavity will yield before the increased pressure they are subject to, and *dilatation* will take place. Hence, according to circumstances, valvular lesions may result in hypertrophy alone, or dilatation alone, or both together; and the last condition is the most common.

When the valvular lesion is so slight as to be adequately met by the reserve power of the heart, or where hypertrophy has developed sufficiently to overcome the difficulty, the lesion is said to be *compensated;* in the converse condition, when the heart is giving way before the lesion and dilatation is in excess, the disease is said to be *uncompensated.*

Hypertrophy and dilatation affect primarily the walls of the cavity immediately behind (in the course of the circulation) the valve which is diseased—that is, the left ventricle is first affected in disease of the aortic valves, and the left auricle in obstruction of the mitral orifice; but the changes are not limited to these cavities respectively. For, first, by continued dilatation of the left ventricle the mitral orifice will become enlarged, and regurgitation will take place in the way above mentioned; and then the left auricle will be placed in the same position of overstrain with regard to the mitral orifice as was the left ventricle with regard to the aortic orifice. And, secondly, without actual dilatation, the pressure in the circulation behind the first affected cavity will be so increased as to involve successively not only the cavities of the heart, but also other structures through which the current of the blood passes. In this way disease of the aortic valves may be followed successively

by dilatation and hypertrophy of the left ventricle, the left auricle, the right ventricle, and the right auricle; and constriction of the mitral orifice successively by similar changes in the left auricle, the right ventricle, and the right auricle.

Congestion of the lungs, which lie, as it were, between the left auricle and the right ventricle, contributes to the effect upon the right side of the heart. It increases the pressure in the pulmonary artery, which the right ventricle may for a time overcome, but ultimately the wall of this cavity yields, and the tricuspid valve becomes inefficient. This may happen either from dilatation of the orifice itself, or from the distension of the ventricular walls drawing down, away from the plane of closure, the segments of the tricuspid valve which are attached to them by the chordæ tendineæ and the papillary muscles. The delay is then felt in the right auricle, and since this receives the blood of the superior and inferior venæ cavæ, the whole of the systemic venous circulation is thereby affected.

The size and shape of the heart are much altered by these changes, and differently according to the cavity or cavities concerned. When the *right ventricle* is much dilated, the triangular shape of the heart is lost, it becomes more globular, and the apex of the heart is formed partly by the right ventricle instead of being formed entirely by the left. With hypertrophy of the *left ventricle*, the heart is enlarged downwards, and the apex is displaced in that direction; but when dilatation is in excess of hypertrophy, there is also great enlargement laterally, and the impulse may be felt, during life, four or five inches to the left of its normal position. The walls of the left and right ventricles may reach double their normal thickness; but the walls of the auricles, though dilating considerably, do not become thick in proportion. As a result of these changes the weight of the heart may increase from the nine, ten, or eleven ounces which are normal, to sixteen, eighteen, or twenty, according to the number of the cavities involved and the extent of hypertrophy. The greatest enlargement occurs in aortic disease, in which the heart has been known to attain the weight of forty-eight ounces. Such examples are known as *bovine* hearts.

Fibroid and fatty degenerations of the myocardium are often the result of chronic valvular disease.

Effects upon other Organs.—The secondary remote effects of valvular disease on other organs, produced as they are by defects in the circulation of the blood which pervades all parts of the body, are numerous and widespread. They are most manifest in the subcutaneous tissues, which become œdematous, and in the lungs, liver, kidneys, spleen, and gastro-intestinal mucous membranes. The most frequent of these are *venous congestion* and *œdema*.

Anasarca.—A general œdema of the subcutaneous tissues is called anasarca. In heart disease it begins in the feet and ankles, and gradually extends up the legs. But even when it is very extensive it is generally confined to the lower extremities and lower half of the trunk, leaving the face, chest, arms, and hands of their normal size.

Lungs.—The delay in the circulation which results from impaired action of the valves on the left side of the heart first affects the lungs; the blood in the pulmonary veins flows with difficulty into the left auricle, and more or less stagnation of the blood in the capillaries takes place. In early stages there is simply undue fulness of the venous radicles in the lung; there is often, in addition, a transudation of serum into the air-vesicles and minute bronchial tubes, so that on section of the lung a quantity of yellowish or almost colourless frothy liquid flows from it; and in advanced cases the most affected parts of the lung become solid, tough, airless, dull red in colour, and uniformly smooth, resembling somewhat the cut surface of the spleen. This condition has been called *splenisation, heart-lung,* or *red induration,* and appears to be a mixed condition of œdema and congestion with minute hæmorrhages. In a later stage, the colour is browner from the presence of pigment (*brown induration*). Both induration and ordinary œdema affect especially the bases of the lower lobes. As a result of local interference with the circulation, some transudation of fluid into the pleural cavity (hydrothorax) often occurs, and there is more or less proneness to inflammatory lesions of the lung, either in the form of bronchitis, pneumonia, or pleurisy.

Liver.—The hepatic vein opens into the inferior vena cava so close to the right ventricle that the influence of cardiac disease upon the circulation of the liver can be readily understood. The organ enlarges considerably, and becomes darker in colour, and in advanced conditions acquires a peculiar appearance of red, yellow, and white mottling, to which the name of *nutmeg liver* has been applied. On section the centre of each lobule is seen to be occupied by the enlarged hepatic vein-rootlet transversely divided, and the adjacent central zone of the lobule is dark-red or purple; outside this is a zone of yellow colour from the retention of bile within it; while the external zone of the lobule is of white or gray colour, which the microscope shows to consist of cells in a state of advanced fatty degeneration.

Kidneys.—These are, as a rule, simply congested, becoming in consequence larger and dark-coloured; but from long-continued congestion a certain amount of fibrous tissue may develop, and by its irregular distribution and contraction may produce a granular condition of the surface.

Other Abdominal Organs.—The *spleen* becomes hard and darker than normal, and, though varying in size, is often smaller. The congestion of the *stomach* and *intestine,* like that of the spleen, is, of course, secondary to that of the liver, since the veins derived from these organs empty themselves into the portal vein. The mucous membrane becomes congested, and after death considerable distension of the vessels, and sometimes hæmorrhages into the substance of the mucous membrane, may be seen. *Ascites,* or dropsy of the peritoneal cavity, is another result of the obstruction to the circulation in the radicles of the portal vein.

Since malignant or ulcerative endocarditis is frequently grafted upon chronic valvular disease, the different organs may be the seat of embolic *infarcts* (*see* pp. 580, 583). The lungs, moreover, may contain infarcts in the later stages of heart disease, independent of the acute form of endocarditis. They occur as conical blood-coloured masses, triangular on section, with the base to the surface, and they occupy commonly the lower lobes, and especially their lower edges. They appear at first sight as if they were simply hæmorrhages into the substance of the lung, and accordingly they have been described as *pulmonary hæmorrhage;* but they are undoubtedly hæmorrhagic infarcts, determined by the impaction in the arteries of fibrinous masses, which have been formed from the blood in the recesses of a dilated right ventricle or right auricle. Commonly about an inch in diameter, these infarcts may sometimes reach a much larger size (*see* Embolism).

Physical Signs.—These are: (1) modifications of the heart sounds, as well as *murmurs* or *bruits;* (2) the evidence derived from inspection, palpation, and percussion as to the *size* and *shape* of *the heart;* (3) the signs of secondary structural changes in other parts of the body. The first of these will be now considered: the second will be discussed mainly under the head of Hypertrophy and Dilatation.

It has been already (*see* p. 566) shown how murmurs in general may arise, during any of the heart's movements, and at each one of its orifices.

It must now be observed that at certain orifices the obstructive murmurs are systolic, and the regurgitant are diastolic, while at the other orifices the regurgitant murmurs are systolic, and the obstructive are diastolic.

Thus, an obstruction at the aortic orifice gives rise to a murmur during the contraction of the ventricle, which forces blood past the obstruction; hence it is the *systolic* murmur (Fig. 56, between 1 and 2).

Incompetence of the aortic valves gives rise to a murmur during the dilatation of the ventricle, because when the ventricle ceases to contract, the aorta recoils upon the column of blood within it, and forces it against and partly through the now incompetent sigmoid valves; hence there is a *diastolic* murmur (Fig. 56, between 2 and 1).

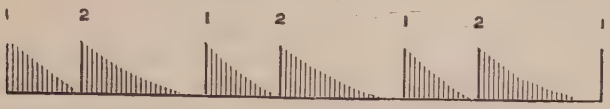
Incompetence of the mitral valve causes a murmur during the contraction of the ventricle, because it is during systole that these valves are called into play; hence with their failure a *systolic* murmur results (Figs. 58 and 59, between 1 and 2).

Obstruction at the mitral valve causes a murmur during the dilatation of the ventricle, because during this dilatation only can a current of blood pass from the auricle into the ventricle. Hence, such a murmur must be *diastolic* in the sense that it takes place during ventricular dilatation. But it may be early, mid, or late.

Frequently the murmur of mitral obstruction is late diastolic or

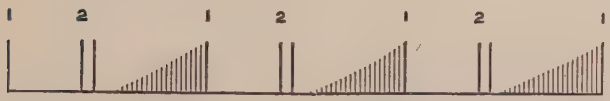
presystolic (Fig. 57, between 2 and 1). This appears to result from the manner in which the auricle contracts. In the normal heart when the ventricle contracts the mitral valves close, and the auricle begins to dilate as blood pours into it from the pulmonary veins; but when the ventricle begins to dilate, the auricle does not

FIG. 56



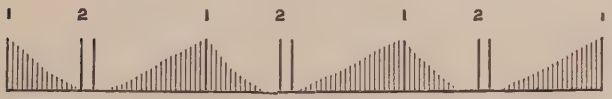
Aortic Disease. Systolic and Diastolic Murmurs.

FIG. 57



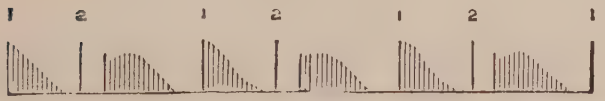
Mitral Disease. Presystolic (late Diastolic) Murmur, with Reduplicated Second Sound.

FIG. 58



Mitral Disease. Presystolic and Systolic Murmurs, with Reduplicated Second Sound.

FIG. 59



Mitral Disease. Systolic and Mid-Diastolic Murmurs, with Reduplicated Second Sound.

In the above diagrams Nos. 1 and 2 represent the first and second sounds respectively, and the shaded lines between them show the periods of time occupied by different murmurs.

thereupon contract, but both cavities are for a time in a state of dilatation; and this is in the first half of *diastole*. Then the auricle, being completely filled, contracts during the latter part of diastole, and its contraction is immediately followed by that of the ventricle. If an obstruction exists at the mitral orifice, it will be especially during the *auricular* contraction that vibration and sound will be produced by the greater force of the current at that time; and hence the murmur produced will occur just before ventricular systole, and will be *presystolic*. Because it is believed to occur during the contraction of the auricle, it has also been called *auricular systolic*. This murmur is further characterised by a peculiar rough, churning, or rumbling quality, and by its becoming louder and louder, until it

terminates in a very loud first sound. It is sometimes followed by a systolic murmur, and this combination is represented diagrammatically in Fig. 58.

A *mid-diastolic* murmur is one which begins just after the second sound, and ends appreciably before the next first sound. Often there is a long interval between it and the first sound; sometimes the interval is very short, especially if the heart is beating quickly, but the murmur even then does not get louder towards the end, like the presystolic. It is either blowing or rumbling in character (Fig. 59, between 2 and 1), and is very frequently accompanied by a systolic murmur of regurgitation. There is thus a combination of a short systolic murmur, a double second sound, and then a long mid-diastolic murmur not running up to the succeeding first sound, resulting in a triple sound which may be imitated by the words "feu-tĕ-feu."

An *early diastolic* is the least frequent of the murmurs of mitral obstruction; it is blowing in character, generally of short duration, and is generally preceded by a definite murmur of regurgitation. Its position in the cardiac rhythm is that of an aortic regurgitant murmur, and it may be represented by Fig. 56, between 2 and 1.

The explanation of the early and mid-diastolic murmurs of mitral obstruction is not so simple as that of the late, or presystolic. The former may take place during the active dilatation of the ventricle; the latter during the passive flow of the blood from the auricle to the ventricle. There is no difficulty in believing this, since the passive flow of blood in the jugular vein is competent to produce a very loud sound, the *bruit de diable*, or venous hum; but it might be thought that if the passive flow of blood produced a murmur in the middle of diastole, the contraction of the auricle ought in the same heart to produce a well-marked presystolic immediately continuous with it. The absence of such a continuation into the presystolic time may, perhaps, be explained by the fact that in such cases the auricle is generally much dilated and weakened.

An early diastolic following upon a mitral regurgitant murmur, suggests that mitral regurgitation unduly fills the left auricle, which on the cessation of systole, returns its excess at once into the ventricle, the auricular flow afterwards continuing silently.

What is here stated of the aortic and mitral valves may be said, *mutatis mutandis*, of the pulmonary and tricuspid valves.

The relation of the murmurs to the rhythm of the heart and the flow of blood through it may be tabulated as follows:

Orifice.	Lesion.	Murmur.	
Aortic or Pulmonary.	Obstruction.	Systolic.	
	Regurgitation.	Diastolic.	
Mitral or Tricuspid.	Obstruction.	Diastolic.	Early. Mid. Late (Presystolic).
	Regurgitation.	Systolic.	

Of these, the pulmonary regurgitant and tricuspid obstructive murmurs are very rare; and murmurs actually due to pulmonary obstruction are less frequent than the remaining five, although a systolic murmur over the region of the pulmonary artery is quite common in association with changes in the quality or quantity of the blood, and is known as a hæmic or functional murmur (*see* Anæmia).

Obviously, the eight possible lesions above indicated (obstruction and regurgitation at each of the four orifices) cannot be distinguished solely by the relation of their murmurs to the sounds of the heart. But we find the means of discrimination in the different points of the præcordial area at which the several murmurs are best heard; and these are determined not so much by the actual position of the valve below the surface as by the direction of the current of blood which is flowing past the orifice at the time, and in which, indeed, the sound-vibrations are largely or entirely produced. Indeed, three of the orifices (aortic, mitral, and tricuspid) lie so close together that if the murmurs were heard only at the orifice concerned, it would be very difficult to distinguish the different kinds. But the flow of blood in the aorta from mid-sternum towards the right clavicle, in the pulmonary artery from the sternum upwards towards the left, and in the heart from auricle to ventricle, conveys each murmur along a special path; and the reflux of blood through the aortic valves into the ventricle, and through the mitral valves into the auricle, acts in a similar way in the case of regurgitant murmurs. The term *area* (mitral area, aortic area) is often applied to the part of the præcordia or adjacent chest-wall where a particular murmur is commonly heard, and in auscultating the heart for valvular disease these areas must be successively examined.

Aortic obstructive murmurs are heard with greatest intensity at the junction of the third right costal cartilage with the sternum, and at the extremity of the second right intercostal space; they can be traced upwards towards the inner half of the right clavicle, and into the vessels of the neck, and they are sometimes heard in the right supraspinous fossa.

Aortic regurgitant murmurs are heard with greatest intensity over the sternum, at the level of the third costal cartilages, and are traceable down the sternum, often to the base of the ensiform cartilage; sometimes also downwards to the left, in the direction of the apex of the heart. More rarely than aortic stenotic murmurs, they may be heard in the back.

When these two murmurs are combined they may be heard together over the junction of the right third costal cartilage with the sternum; sometimes they are only heard separately, and then respectively above and below this point.

Mitral obstructive murmurs are heard most loudly at the point of impulse of the heart against the chest; though sometimes audible more or less imperfectly between this point and the sternum, they

are always best heard at this spot, and are often strictly limited to an area of an inch or an inch and a half in diameter. The stethoscope should always be placed over the actual heart-beat, as found by examination, and not only over the spot where the impulse should be normally found.

Mitral regurgitant murmurs are mostly heard with greatest intensity at the apex of the heart, but they are commonly widely diffused, slightly over the præcordial region, towards the sternum and the base of the heart, and also outwards to the left. In the axilla they often lose in loudness, but are again heard at the angle of the left scapula, and even all over the base of the left chest, and over the base of the right chest; sometimes, at least on the left side, as loudly as in front.

Pulmonary obstructive murmurs are heard with great intensity in the second left intercostal space at its inner end, and can be traced outwards in that space, and upwards towards the left clavicle.

Pulmonary regurgitant murmurs are heard at the junction of the third left costal cartilage with the sternum, and thence downwards over the right ventricle, along the left border of the sternum.

Tricuspid obstructive murmurs have been heard, with a presystolic or mid-diastolic rhythm (like mitral obstructive murmurs) at the left side of the sternum, over its junction with the fourth costal cartilage.

Tricuspid regurgitant murmurs are heard at the lower half of the sternum, over an area corresponding pretty closely to the part of the heart left exposed between the two lungs; but they are sometimes limited to the base of the ensiform appendix, sometimes extend to the right nipple, and are probably heard at the apex when dilatation carries the right ventricle in that direction.

In applying the above rules to any cases before us we must bear in mind the following facts: (1) Valvular disease may exist without a murmur being produced. (2) Very loud murmurs are heard over a large extent, and so encroach on the areas of healthy valves. (3) Two or more valves may be diseased at the same time, as, for instance, the aortic and mitral together; the mitral and tricuspid together; or even the aortic, mitral, and tricuspid at the same time. (4) Besides valvular disease many other conditions (aneurysm, anæmia, and certain diseases of the lungs and pleura) give rise to murmurs over the præcordia.

In the presence of these valvular murmurs, the natural heart-sounds may be considerably altered or partly disappear. Since the second sound of the heart is due to the closure and vibration of the sigmoid valves, disease of these valves resulting in their imperfect closure must be followed by a diminution of the loudness, or by complete abolition of the second sound corresponding to the diseased orifice, so that with a regurgitant aortic murmur the second sound is enfeebled or absent. The same applies to the mitral valve; its perfect closure contributes to the first sound, which must be weakened when regurgitation is present. Since both sides of the

heart contribute to the natural sounds, the disease of one side alone may fail to abolish them. Another frequent modification in heart disease is accentuation of the second sound at the pulmonary orifice; this occurs especially in mitral regurgitation, and is brought about by the increased tension in the left auricle, pulmonary veins, and pulmonary circulation generally. Mitral constriction in early stages is accompanied by an accentuated first sound at the apex, and a reduplication of the second sound at the base, or lower sternum.

Thrills.—The thrill, or *frémissement cataire*, is only an occasional physical sign of valvular lesion. It is never present without a murmur, and is, indeed, due to the fact that some of the vibrations which cause the sound are of a nature to be felt also. It is most common in mitral constriction, and accompanies a large proportion of presystolic (or late diastolic) murmurs, and some mid-diastolic murmurs. Thrills with other murmurs are much less common, and the valvular regions which they accompany may be arranged in the following order of frequency: pulmonary stenosis (congenital), tricuspid regurgitation, aortic stenosis, aortic regurgitation, mitral regurgitation, tricuspid stenosis.

Symptoms.—In describing these, it will be best at first to limit ourselves to disease of the valves on the left side, the aortic and mitral valves. Pulmonary valve disease is rare except in the congenital form, which will be considered hereafter, and tricuspid disease is mostly secondary to mitral affections.

Disease of the aortic and mitral orifices may exist practically without any symptom for long periods, and may be detected only on auscultation. This is explained either by the slightness of the lesion or by the hypertrophy of the muscular walls being sufficient to meet the additional strain upon them, so that the valvular lesion is *compensated*. But where the compensation is insufficient, or where hypertrophy is accompanied by dilatation, symptoms sooner or later manifest themselves.

Mitral disease may be taken first, as being the most frequent. The symptoms are in many points the same, whether the disease be obstructive or regurgitant. The early symptoms are pain or distress at the heart, palpitation, shortness of breath, and swelling of the feet; in later stages, evidences of failing circulation in the various organs of the body. Congestion of the lungs is shown by cough, by mucous expectoration, by occasional hæmoptysis, which may arise from the pulmonary infarcts above described, by orthopnœa at night or continually, and by dyspnœa on the slightest exertion. On examination crepitations will be heard at the bases of the lungs, and in advanced cases dulness, with deficient vesicular murmur, and deficient tactile vibration. General venous stagnation is shown by a rich red colour or actual lividity of the lips, cheeks, ears, and extremities, and by the occurrence of anasarca. The congested liver is large and smooth, reaching perhaps to the level of the umbilicus; and the skin is slightly jaundiced, the yellow tinge of the forehead combining with the deep red of the lips and cheeks

to give a very characteristic appearance to the sufferer. Other results of the hepatic stagnation are ascites, congestion of the spleen, and more or less frequent vomiting. The secretion of the kidneys is also affected, the urine being scanty from low arterial pressure, reduced perhaps to ten or fifteen ounces daily, high-coloured, depositing large amounts of urates and containing albumin and fibrinous casts; the quantity of albumin is generally small, and varies inversely as the efficiency of the heart. Drowsiness or restlessness, and in advanced cases occasionally delirium, show the effect upon the circulation of the brain. Death takes place ultimately from cardiac failure, from œdema of the lungs, from sloughing of the skin and exhaustion, or from malignant endocarditis, or other complication.

The differences between mitral regurgitation and constriction are mainly seen in their effects upon the heart and the vessels, and in the average time they take to develop symptoms.

Mitral Regurgitation.—The left ventricle becomes dilated and hypertrophied; the physical signs are the displacement of the impulse downwards and outwards, the systolic murmur audible at the apex in the axilla, and at one or both bases behind, and the accentuation of the second sound over the pulmonary artery. In early stages the impulse is more or less heaving and regular, the pulse soft, with a high percussion wave, a rapid fall, a feebly developed tidal wave, and moderate dicrotism. In later stages the heart becomes irregular, with a more and more feeble impulse, while the pulse is correspondingly small, feeble, irregular, and compressible, and its sphygmographic tracing shows a respiratory wave (*see* p. 570).

Mitral constriction at first affects rather the left auricle than the left ventricle. The former is hypertrophied in its endeavours to overcome the obstruction, and the latter remains of its normal size. Thus in early stages the impulse may be in its usual situation, and the condition is revealed only by the characteristic presystolic murmur (*see* p. 593), which is often followed by a reduplicated second sound. Where the murmur is best heard there is often a palpable vibration, or thrill, which is, like the murmur, presystolic in rhythm.

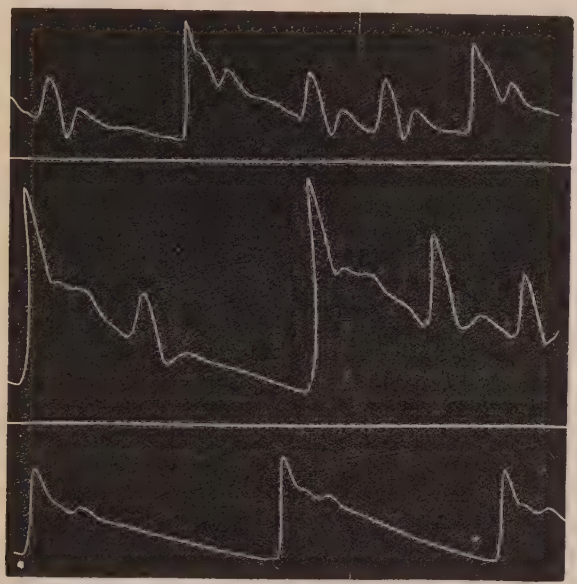
In later stages mitral constriction is apt to be complicated by either tricuspid or mitral regurgitation; the presystolic murmur is less often observed, being replaced by the combination previously described (*see* p. 594). Sometimes this and the presystolic alternate on succeeding days in the same case. Exceptionally, the murmur may be early diastolic. Sometimes mitral constriction is unaccompanied by any murmur at all; and not infrequently there is no diastolic murmur, but a reduplicated second sound and a short systolic murmur, inaudible behind, of which it is not always easy to say whether it is a murmur of mitral regurgitation or not.

In the latest stages of cases of mitral constriction the heart's action is most irregular and tumultuous. The rhythm of the

diastolic sound is incapable of recognition, and there is a continuous rumble or roaring sound with a systolic murmur interposed at intervals.

The pulse in early stages may be absolutely normal, of medium pressure, and the heart regular; but in later stages irregularity is

FIG. 60



A. Mitral Regurgitation, complicated by Renal Disease: Irregular Heart with Premature Beats. Pressure, Six Ounces.
B. Mitral Constriction: Irregular Heart with Premature Beats. Pressure, Three Ounces.
C. Pulse of Mitral Constriction under Treatment. Pressure, One and a Half Ounces.

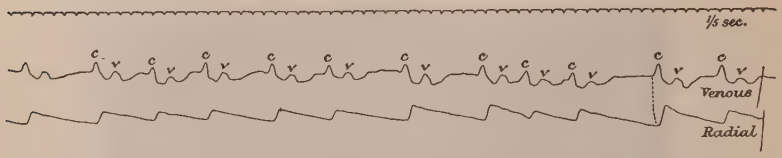
the characteristic feature, and it often happens that at every fourth or fifth beat the pulse expands twice close together—*i.e.*, it receives a second wave of blood from the heart before the first has been passed over to the arterioles (premature systole). Tracings of pulses in mitral disease are shown in Fig. 60.

In the late stages of mitral stenosis, as well as in myocardial degeneration from other causes, the auricle is distended; and instead of undergoing its normal rhythmical contraction, it is the subject of fine, disorderly, tremulous contractions, a condition described as *auricular fibrillation*. The auricular contractions are transmitted to the ventricle, and result in complete irregularity of the beats of the heart and pulse. Tracings of this condition are shown in Fig. 61.

Mitral constriction often gives rise to hæmoptysis quite early, and

is perhaps more often than mitral regurgitation the cause of hemiplegia from embolism of the cerebral arteries. As to their relative duration and fatality it is difficult to speak, because while the majority of cases of regurgitation arise out of rheumatic fever, many cases of constriction give no previous history by which the

FIG. 61



A Polygraphic Record from a Patient with Complete Irregularity of the Heart. Each systole of the ventricle is accompanied by *c* and *v* waves in the upper or venous curve. There is a total absence of the usual presystolic wave *a*. The absence of *a* and the presence of the irregularity are attributed to fibrillation of the auricle. (*After Lewis.*)

onset can be fixed; and further, they often co-exist, either one being secondary to the other. Some cases of regurgitation last very many years, and probably few cases of mitral constriction last so long. In either case life may be cut short by the occurrence of septic changes (malignant endocarditis).

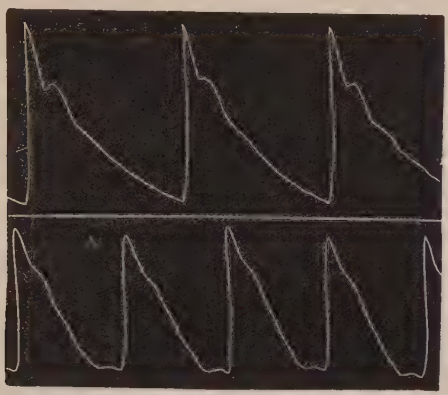
When obstruction and regurgitation are associated together at the mitral orifice, they give rise to various combinations of systolic and diastolic murmurs, of which some have been already mentioned (*see also* Figs. 57, 58, 59).

Aortic Disease.—The most common form of aortic disease is that in which evidence of both obstruction and regurgitation is present. Next most common is regurgitation alone, and pure aortic obstruction is relatively rare. Aortic valve disease arises much less frequently than mitral valve disease from rheumatic fever, and is often the result of chronic inflammatory and degenerative changes in the valves, and in the aortic wall—arteritis and arterio-sclerosis. Syphilis is an important factor in the production of aortitis, and there are other causes, which are discussed elsewhere (*see* p. 644). Such causes are assisted by frequent and continued strains upon the circulation, especially from the excessive use of the arms, such as arises in blacksmiths, sawyers, and others with laborious occupations. Sudden rupture of the valves also sometimes takes place.

Regurgitant cases are characterised by the murmur already described (*see* pp. 592, 595). But occasionally no murmur is audible; and rarely, sometimes with, and at others without the typical murmur of aortic regurgitation, a rumbling presystolic murmur is heard at the apex, although the mitral valve is perfectly healthy. This fact was first described by Flint (*see also* p. 606). The heart is commonly dilated and hypertrophied, and the impulse is carried downwards and slightly outwards. The effect on the pulse is striking and characteristic; the hypertrophied ventricle drives the

blood with great force into the arteries, causing a high percussion wave, but, as the valves close imperfectly, the column of blood is not sustained, and the dicrotic wave is badly developed from the slightness of the recoil (Fig. 62). The sudden rise of the pulse-wave, and its equally sudden subsidence, yield a peculiar sensation

FIG. 62



A. Pulse of Aortic Regurgitation. Pressure, Three Ounces.
B. Pulse of Aortic Regurgitation. Pressure, Four and a Half Ounces.

to the finger, which is expressed by the various names given to this form of pulse, such as *kicking*, *splashing*, *water-hammer*, and *shotty*. The terms *refluent* and *collapsing* refer to the sudden subsidence of the wave, which is, indeed, its essential feature. All over the body the sudden and extensive movements of expansion and contraction in the arteries produce marked effects. The vessels of the neck throb visibly and often painfully; the digital arteries can be felt with unusual distinctness, and the pulsation of the retinal arteries can be easily seen with the ophthalmoscope. Aortic incompetence may also cause *capillary pulsation*. This may be seen under the nails, in the cheeks, or in the area of dilated capillaries produced by drawing a sharp point over the surface of the forehead; or by pressing a microscope glass-slide upon the mucous membrane of the everted lower lip. In either case the vascular area under observation becomes alternately darker and paler with each beat of the heart.

Arterial Sounds.—Auscultation of the arteries reveals some differences from the normal. In the carotid and subclavian the usual second sound is lost, or is rarely replaced by a murmur. In the other arteries, normally silent, there is a systolic sound. In the femoral there is often a double sound; but the most interesting fact is that a double murmur can be obtained on pressure with the stethoscope, the first element of which is the usual direct pressure murmur (*see* p. 578), and the second is a pressure murmur due to the reflux of blood caused by the aortic valvular defect.

Patients with aortic regurgitation are often markedly *anæmic*, with pale face and lips, and mucous membranes; but when, in the course of time, secondary mitral regurgitation takes place, the blood in the venous system stagnates, and congestion of the lips and cheeks replaces the former bloodlessness. Shortness of breath, cough, and mucous expectoration, swelling of the feet, pain at the upper part of the sternum, especially on exertion, scanty urine, and albuminuria are the common accompaniments of this disease, and a termination by sudden syncope is much more frequent than in other forms of valvular disorder.

In pure *aortic stenosis* the murmur is systolic, audible in the second right intercostal space near the sternum, traceable up towards the right clavicle, and audible in the carotid arteries. If stenosis is considerable a thrill may be present, felt at the same spot, and also systolic. The heart will be hypertrophied or dilated, in proportion to the amount and duration of the obstruction. The pulse is often characteristic; the obstacle interposed in the current of blood prevents the full effect of the ventricular contraction upon the column of blood in the systemic arteries, and the pulse can be felt to have lost its suddenness and to rise quite slowly. The pulse tracing is then *anacrotic*, that is, the percussion wave is lower than the succeeding tidal wave, and appears as an elevation on the ascending limb. In the extreme variety of this form this wave is rounded off, or entirely absent, and the tracing resembles that shown in Fig. 66, A (p. 646). In some cases the tracing has a tidal wave as high as the percussion wave, with a deep notch between them (*pulsus bisferiens*): this is ascribed by some to instrumental defects.

The symptoms of combined aortic obstruction and regurgitation, or double aortic disease as it is often called, are mainly those due to regurgitation. A systolic and a diastolic murmur are heard, forming the *to-and-fro* murmur, and the patient is commonly anæmic in the early stages; later on, the left ventricle becomes more dilated, the mitral orifice widens, the valve becomes insufficient, and the symptoms due to this condition show themselves.

Tricuspid Regurgitation.—On the right side of the heart the only form of valve disease that is at all common is *tricuspid incompetence* leading to regurgitation; and this is mostly due to distension of the right ventricle, causing enlargement of the tricuspid orifice, which the cusps of the valve are unable to cover; and only rarely to primary disease of the valve itself. Such dilatation of the right ventricle may arise from any increased pressure in the ventricle and pulmonary artery, whether from chronic disease of the lungs (emphysema, bronchiectasis, very chronic phthisis) or from incompetence or obstruction at the mitral valve. It is thus frequently associated with mitral disease and the other forms of left side failure. It is commonly accompanied by the evidence of dilatation of the right heart, and by the various degrees of œdema, anasarca, and venous congestion which indicate a difficulty in the return of blood to the right heart and lungs. These have already been

enumerated under the later symptoms of mitral disease. The signs more characteristic of tricuspid regurgitation are, the systolic murmur, and the jugular and hepatic venous pulsations. The murmur of tricuspid regurgitation has been already described (*see* p. 596): it is sometimes accompanied by a systolic thrill over the lower end of the sternum. Pulsation in the internal jugular veins is seen as an extensive undulating movement of elevation and subsidence at the side of the neck between the ear and the clavicle posterior to the course of the carotid artery. The external jugular may pulsate at the same time. A tracing shows that the auricular wave is suppressed from paralysis of the auricle; and a large wave (*ventricular*) occupies the sphygmic period, which is due to the right ventricle acting through the leaking tricuspid orifice upon the blood in a distended and ineffective auricle.

In tricuspid regurgitation also the force of the right ventricular contraction may be transmitted to the hepatic veins, so as to cause *hepatic venous pulse*, or *pulsating liver*. The organ is commonly much enlarged, and can be felt throbbing over its whole surface; and the pulsation is sometimes even conveyed behind into the right loin under the last rib, so that the liver can be felt to expand between the hands placed in front and behind.

Tracings may be obtained which resemble those from the jugular vein in the same circumstances.

Tricuspid obstruction (*see* p. 596) is less common, and is generally observed in conjunction with other valve disease. No special group of symptoms can be referred to it apart from those seen in tricuspid regurgitation.

Disease of the pulmonary valves is mostly congenital if of old standing; and if acute it is the result of malignant endocarditis. *Pulmonary obstruction* is the usual condition in the former case: the valves are united together to form a cone, with only a small opening for the passage of blood, but they may close perfectly. The murmur is systolic (*see* p. 596), and is often accompanied by a marked systolic thrill over the same area. The obstruction leads to dilatation of the right ventricle, and at the same time so far hinders access to the lungs that a very imperfectly aerated blood circulates, and the patient is habitually cyanosed (*see* Congenital Malformations).

Pulmonary regurgitation sometimes occurs as a result of mitral stenosis, the valves yielding before the great pressure in the pulmonary artery; a diastolic murmur is heard along the left side of the sternum.

Malignant endocarditis may cause a double (systolic and diastolic) murmur at the pulmonary orifice like that of aortic disease, the respective murmurs having the position previously noted. The symptoms under such conditions have already been detailed (*see* Malignant Endocarditis).

Complications.—Every result of simple venous stagnation must be considered a part of heart disease, such as red and brown

indurations of the lung and albuminuria. But pleurisy, pneumonia, embolism, and malignant endocarditis may be regarded rather as complications. With a failing circulation fibrin may be deposited upon the valves and in recesses of the dilated cardiac cavities. Hence detached particles may be carried into the cerebral vessels, causing hemiplegia; into the splenic or renal vessels, producing the characteristic infarcts, or more rarely into the vessels of the limbs, abolishing the pulse or leading to gangrene. Malignant endocarditis is frequently grafted upon chronic disease.

Diagnosis.—In the diagnosis of valvular heart disease many questions have to be considered. It has to be determined (1) whether a murmur is due to valve lesion or to some other cause, endocardial or exocardial, the former including change in the muscular walls; (2) at which orifice it arises, and if there are two, whether one is dependent on the other; and (3) what are the conditions of the several cavities of the heart, and how they are affected by whatever valvular lesion exists. Very important information as to the heart's position, action, and valvular working can be got by the eye and the hand; and these should always be used in conjunction with the stethoscope. The Röntgen rays will also assist in the estimation of changes in the size and shape of the heart's cavities. (*See* Plate VI.)

(1) The murmurs of valve disease are apt to be confounded with those due to other conditions. *Anæmia* produces a harsh systolic murmur over the pulmonary area. Seeing the rarity of organic pulmonary disease, this is mostly distinctive enough, but with very considerable anæmia murmurs extend over the whole præcordial area, arising, no doubt, at other orifices besides the pulmonary. As in such cases the patients are short of breath, with tendency to palpitation and to swelling of the feet, the diagnosis may be difficult. The marked pallor of the anæmic cases, the absence of history of rheumatism, or other precursor of heart disease, and the diminution of the murmur under the use of ferruginous tonics, are points which will help. But anæmia may also undoubtedly itself be a cause of mitral regurgitation; the deficient quality of the blood causes malnutrition of the wall of the ventricle; this dilates, the mitral orifice yields, and regurgitation is the result. This is, indeed, an actual lesion, and the murmur is immediately due to structural changes in the orifice, if not in the valve itself; but inasmuch as they are primarily due to a condition of the blood which, together with its results, is curable, this murmur is often spoken of as *functional* or as *hæmic*. The diagnosis must, at any rate, be made between this and chronic valvular disease, and it can generally be effected by a consideration of the preceding and associated circumstances—viz., the absence of rheumatism and the decided anæmia.

Aneurysm of the aorta frequently gives rise to a murmur at the base of the heart which may be mistaken for that of aortic obstruction. Indeed, a simple systolic murmur in the aortic area, unaccom-

PLATE VI

Skiagram of a case of mitral stenosis in a child of nine, showing the rounded form of the heart due to enlargement of the right side, and of the left auricle. (Taken by Dr. A. C. Jordan.)

[*To face p.* 604.

panied by regurgitant murmurs, is much more often due to aneurysm than to stenosis of the valves. Abnormal pulsation to the right of the sternum, and increased area of dulness, should be sought for as further evidence. If the murmur is localised at a point not strictly corresponding to the known areas of valve disease, aneurysm is still more probable.

Pericarditis often gives rise to a to-and-fro sound, very like the murmur of double aortic disease. It is, however, usually rougher, less uniform in loudness over a large area, not strictly localised to the usual area of aortic disease, and perhaps here and there not strictly synchronous with the two periods of the heart's beat. A short history of acute illness, unusual pain or distress at the heart, increased area of præcordial dulness in an upward direction, and absence of splashing pulse, point to pericarditis.

Another difficulty arises from sounds, synchronous with the heart's action, produced outside the heart—*exocardial* murmurs. Some of these are due to the heart beating strongly against the lung (*cardio-pulmonary*). The most common of these is a short, high-pitched, systolic murmur, often limited to the apex, which is heard in nervous or excited persons when they are under medical examination. The murmur may be heard at the left scapula, behind as well as in front. A systolic apex murmur, audible only during inspiration, is probably often exocardial. Others are produced by displacement of the heart, as when it is compressed by pleural effusion, or by deformities of the thorax; and others by morbid conditions of the lung and pleura immediately adjacent to the heart, and mostly on the left side. Very extraordinary murmurs are sometimes heard when a large pulmonary cavity is in close contact with the heart, the air being driven suddenly out of the cavity with each cardiac impulse. But the recognition that a murmur is endocardial and produced at a valvular orifice does not carry with it the diagnosis of disease of the valve. Ventricular dilatation not only from anæmia, but from any cause whatever, may lead to an apex systolic murmur; and such an occurrence is most common in Bright's disease, in alcoholism, and in arterio-sclerosis; and acutely in connection with infectious disease.

Chronic renal disease may bring about hypertrophy of the heart, and even dilatation and murmur, and the case will then closely resemble one of mitral disease with secondary albuminuria. The difficulty is increased by the fact that the kidneys in a state of chronic congestion from heart disease may become granular; and that a heart dilated, as a result of extreme arterial tension in renal disease, will cause secondary stagnation in the venous system, like one affected with primary mitral disease. In primary heart disease one must look for the history of rheumatism or other cause of endocarditis. The urine has the characters described (*see* p. 598); and the pulse is small and of low tension. But in renal disease the urine is more likely to be pale, though also scanty, and to have a more uniform quantity of albumin; and the pulse is one of high

tension. In enlargements due to arterio-sclerosis and to alcohol, conditions which are often combined, the arterial tension is variable, and albumin is often absent : the diagnosis may have to depend on the history or associated conditions.

Conversely a valvular lesion is sometimes present when no murmur can be heard: this is most frequently the case in the later stages of mitral constriction, when the strength of the auricle is failing.

(2) The diagnosis of the different forms of valvular disease one from another depends for the most part on the character of the murmurs, and the extent to which they are audible over the præcordial area. A murmur may be conveyed beyond the area of one valve into the area of another, when it will be necessary to compare carefully the intensity of the sound at different points. Aortic regurgitation and mitral regurgitation are nearly always indicated by their characteristic murmurs; though it is doubtful if one can speak positively as to mitral regurgitation unless the murmur is heard behind at the angle of the scapula as well as in front at the apex, murmurs of more limited range being possibly produced within the ventricle itself. Mitral obstruction frequently exists without its characteristic murmur, as above stated. Pure presystolic murmurs, pure diastolic murmurs, and others intermediate in rhythm, when heard at the exact impulse (and not heard at the base), are very strong evidence of mitral obstruction. But murmurs almost identical with these are sometimes heard at the apex in association with aortic regurgitation (*see* p. 600), with adherent pericardium, and with a dilated ventricle under other conditions. The explanations of these anomalies are various: Vibrations of the anterior mitral cusp from impaction upon it of the aortic regurgitant current, or from its being driven in upon the auriculo-ventricular current; mingling of the above two currents; the formation of a *veine fluide* in consequence of the dilatation of the left ventricle, while the mitral orifice is of normal size. None of them is free from difficulties.

(3) The estimation of the condition of the heart as a muscular organ is based upon the physical signs described under Hypertrophy and Dilatation (*see* p. 613), and upon the performance of its functions as shown by the pulses (jugular and radial tracings) and by the effects upon the circulation in general (dropsy, albuminuria, &c.).

No diagnosis can be made without an examination of the heart and lungs; but it is interesting to note that there is often in children and young people a superficial resemblance between mitral disease and phthisis, since the former may produce marked pallor, emaciation, and hæmoptysis.

Prognosis.—The efficiency of the heart as a propelling organ, and not the loudness of the murmur, must be taken as the guide to an estimate of the duration of life. Acute temporary dilatations may be entirely recovered from, but pronounced valvular disease is practically incurable. The most that can be done is to preserve

the efficiency of the organ as long as possible, and this may be to the end of a long life. The object is attained if the lesion is but slight, or if, when the patient is relieved from undue strain of body or mind, and is put under the best possible conditions in every respect, an adequate compensatory hypertrophy is developed. Even under relatively adverse circumstances, with a comparatively severe lesion, the heart may remain *in statu quo* for years. In any given case one should take into consideration the valve affected, the condition of the heart at the time, and the history of the patient's illness. When a patient comes under treatment for the first time, his temporary improvement is highly probable; but frequent relapses argue insufficient compensation, and a short future. The amount of hypertrophy and dilatation compared with the duration of the illness, so far as it can be estimated, will suggest the rate of progress that may be expected; but considerable allowance must be made for the effects of treatment, if it has not been thoroughly instituted. As to the valve affected, something has been already said. Aortic obstruction is least serious, aortic regurgitation most. Mitral obstruction often remains stationary for many years, but much constriction may be very rapidly fatal. Free mitral regurgitation also tends to shorten life considerably. The prognosis is naturally more grave when two or more lesions co-exist; aortic disease rapidly terminates when the mitral valve becomes secondarily involved, and tricuspid regurgitation is a serious complication of left-side disease. Persisting pyrexia should make one suspect malignant endocarditis, when the prognosis becomes at once unfavourable.

Treatment.—The mere presence of an endocardial murmur, the result of old disease, does not call for any special treatment by drugs so long as the cardiac muscle remains unaffected, or is but slightly hypertrophied, and not dilated; so that the heart beats efficiently at the normal rate, and the patient is free from dyspnœa, discomfort, and dropsy. When the lesion is thus *compensated*, all that is necessary is to warn the patient against over-strain and over-exertion, to maintain the nutritive powers of the body, without allowing excess, and in case there is a tendency to deficient nutrition, to meet it by cod-liver oil, iron, and other tonics.

When symptoms definitely show themselves, such as shortness of breath and palpitation, more decided action is required; and the treatment involves three indications which may have to be pursued with more or less vigour, according to the stage or severity of the disease:

(1) *Rest* is the first of these. The patient should be placed in bed, in the recumbent position, if possible. If this is prevented by orthopnœa, the body should be supported by pillows or a bed-rest to take off all possible strain from the muscles, and hence from the circulation. Sudden movements of all kinds should be forbidden, and complete quiet and freedom from anxiety and excitement should be enjoined.

(2) The relief of the circulation by *depletion* in one or other form

is the next indication. Thus a free action of the bowels should be obtained, and in severe cases the more powerful hydragogue cathartics, like elaterium and compound jalap powders, should be employed, and repeated from time to time. The kidneys should be stimulated to act by diuretics, such as acetate and citrate of potassium, nitrous ether, squill, and scoparium; and these may be used even when albuminuria is present, so long as it is clear that the albumin is due only to venous congestion. Under the use of diuretics the urine increases in quantity and the albumin diminishes. Diaphoretics may similarly be employed. If there is much anasarca, the legs may be punctured, or drained by Southey's tubes. Ascites may be tapped, and by both these proceedings the pressure on the circulation is diminished. In severe cases of advanced cardiac disease recourse may be had to direct depletion by *venesection;* and this is of especial service when the right side of the heart has become dilated and so engorged that it is powerless to contract upon its contents, and death is threatened by paralysis of its walls. In such circumstances the heart's action is feeble, irregular, and fluttering; the radial pulse is correspondingly small and compressible; the surface is livid and cyanosed, the lower half of the body œdematous, the large veins prominent, and perhaps pulsating in the neck. The withdrawal of blood to the extent of 10 or 12 ounces by an opening in the basilic or external jugular vein at once relieves the engorged condition of the right ventricle, which can again contract efficiently upon its contents, while time is given for the action of diuretics, purgatives, and the special drugs which will shortly be mentioned.

Closely allied to the subject of depletion is the question of *diet.* This should be sufficient, simple, and readily digestible; it may be mixed solid and liquid in quantity at any one time not to overload the stomach, and of a nature not to cause flatulence or distension.

(3) Certain drugs, usually described as *cardiac tonics,* act by increasing the force of the heart's contraction. Digitalis is the one which has the most powerful influence in restoring the dilated and weakened ventricle to a state of efficiency. Under its use the quick, weak, irregular contractions become slower, stronger, and more regular. The diastolic pause of the heart is lengthened, and time is given in the interval for nutritive repair, and hence a more efficient contraction afterwards. This is a brief statement of the chief influence of digitalis; but its action is really much more complicated. It slows the rate of the heart by vagus-inhibition: and by this gives time for the improvement secondarily of contractility, conductivity, and excitability. It also increases directly the contractility of the heart, making it beat stronger: but in overdoses it diminishes conductivity, and may thereby lead to heart-block (*see* p. 562). In cases of auricular fibrillation its good effects are attributed to its reducing conduction through the auriculo-ventricular bundle. In small doses it diminishes excitability, and will thus lessen the frequency of occurrence of extra-systoles; but

excitability is increased by large doses, and extra-systoles are produced. Digitalis also raises the arterial pressure, as a result in different degrees of increased heart-power, of vaso-constriction in the splanchnic area, and of contraction of peripheral arteries.

Digitalis may be given in powder, infusion, or tincture, or as one of its active principles, digitalin or digitoxin. In serious cases 2 drachms of the infusion or 10 or 15 minims of the tincture may be given every three or four hours at first, and after twelve or twenty-four hours less frequently or in smaller doses. The dose of digitoxin is $\frac{1}{240}$ grain to $\frac{1}{100}$ grain. The usual clinical effect of digitalis is that the heart beats more slowly and powerfully, free diuresis takes place, dropsy and dyspnœa diminish, and the comfort of the patient is secured. But its action requires to be watched; an overdose causes sickness and headache, in addition to the very slow, irregular pulse, perhaps with coupled beats.

Digitalis is not suitable for all conditions of valvular disease; it is contra-indicated in cases of hypertrophy without dilatation, when the heart's beat is powerful, of moderate quickness only, or even slow, and nearly or quite regular. It only increases the violence of contraction in such conditions.

In aortic regurgitation the fact that digitalis prolongs the diastole seems in one respect to be a disadvantage; for since during the diastole the blood flows back from the aorta into the ventricle, the longer the diastole the greater the regurgitation, and the less for the time being is the supply of blood to the brain and other tissues. There would thus be a greater risk of syncope from aortic disease during the use of digitalis; but the tendency is probably as a rule neutralised by the more powerful action of the heart during systole. As a fact, digitalis has generally a smaller field for service in the slower and more forcible action of the heart of aortic disease; but when the ventricle is acting feebly and irregularly, it may be used.

Some other drugs have an action like that of digitalis. The most important of these is strophanthus; of this 10 to 15 minims of the tincture should be given as a dose; it is not so generally trustworthy as digitalis. Convallaria (5 to 30 minims of a 1 in 8 tincture) and caffein citrate (5 to 10 grains) are also employed.

Strychnine also acts as a valuable cardiac tonic, and may be given when the heart's action is feeble or ineffective from any cause; but it has not the special effect on an irregular heart which is shown by digitalis. It is given in doses of 5 or 7 minims of the liquor internally, or 2 to 5 injected subcutaneously. In critical conditions it may be assisted by the stimulant action of brandy, ether, or ammonium carbonate.

In aortic disease with hypertrophy, one of the most distressing symptoms is the violent action of the heart, and the throbbing of the great vessels in the neck and over the body generally; and this may be much relieved by the use of a small dose of tincture of aconite (1 to 3 minims), by bromides, or by a small dose of morphia.

Other symptoms and complications may have to be treated. Pain over the heart is often severe, and may be relieved by belladonna plasters, by small doses of morphia internally, or by subcutaneous injection in some cases; but this drug must be used with very great caution, and not at all in advanced cases with much cyanosis. Cough may be treated with small doses of expectorants and sedatives, and vomiting by effervescing salines. Little can be done for the secondary affections of the lungs. Slight pleural effusion may be treated by the application of tincture of iodine; larger effusions may be tapped. Induration of the lung is not amenable to local treatment; pulmonary hæmorrhage is rarely sufficient to threaten life, and does not require styptics.

Cerebral embolism, again, is beyond the reach of direct treatment, but embolism of an artery in one of the limbs must be met by the application of warmth, to avoid as far as possible the supervention of gangrene. Throughout, the diet should be in moderate quantity, light, easily digestible, and unstimulating. With a much weakened heart, and in late stages, however, stimulants in the form of brandy, whisky, or sherry, will form an essential part of the treatment.

The hill-climbing of Oertel and the baths with graduated exercises employed by Schott, at Nauheim, do not seem so suitable for the treatment of valvular lesions as for other less permanent lesions of the heart, unless in quite early stages. They will be described later (*see* p. 628).

CONGENITAL MALFORMATIONS

Malformations of the heart arise from defects in its development, which is normally not complete until the closure of the ductus arteriosus and foramen ovale some days after birth; and their origin can be only understood from a knowledge of the manner in which development probably proceeds. The organ commences as a straight tube, receiving a single vein behind, and giving off a single arterial tube in front; it subsequently becomes bent on itself, the bent part forming the ventricle. Between this and the single artery is the bulbus cordis, while posterior to the auricle is the sinus venosus. Subsequently, the auricle is divided by a septum and the sinus venosus is fused with the right division. A septum also forms in the ventricular cavity by the down-growth of the right and left ventricles, and the bulbus cordis is merged into the right ventricle, and forms the infundibulum. The growth and development of the bulbus cordis, and the union of the anterior and posterior portions (*endocardial cushions*) of the original auriculo-ventricular orifice, thus dividing the mitral orifice from the tricuspid, assist in completing the septum of the ventricles. The tissues of the endocardial cushion supply the upper membranous part, or *pars membranacea septi*, which lies between the anterior and right posterior aortic valves. The

aorta and pulmonary artery arise by the growth of a septum in the first-formed single arterial tube; and the final change takes place when, in consequence of the diversion of blood to the lungs after birth, the ductus arteriosus and foramen ovale are both closed.

Arrest of this process in any stage will lead to a congenital malformation. It has occurred so early as to leave the heart with only two cavities—an auricle and a ventricle; or with three cavities —a ventricle and two auricles. But these are very rare cases, and the children mostly live but a short time after birth. One of the commonest lesions is stenosis of the outlet from the right ventricle, either by union of the pulmonary valves, or by a constriction of the ring just below them, or of the infundibulum itself, or by an imperfect septum dividing the infundibulum from the ventricle. Such conditions are regarded by Keith as invariably due to defects in development or to want of expansion of the bulbus cordis, and never to intra-uterine endocarditis. If the outlet of the right ventricle is thus obstructed in early fœtal life, the pressure in that cavity is relieved by overflow into the left ventricle, through the still unclosed septum: and this opening then becomes permanent. According to the stage of development at which the arrest has taken place, the deficiency may be a very large one, or a mere perforation in the upper part; and in this latter case the aperture occupies the *pars membranacea*. When the deficiency is a large one, the aorta frequently arises from the right ventricle, or from both right and left ventricles, and the foramen ovale and ductus arteriosus may one or both be pervious. If the constriction of the pulmonary outlet takes place after the septum of the ventricles is complete, either the foramen ovale or the ductus arteriosus must remain pervious.

Constriction or obliteration of the aortic orifice or of one auriculo-ventricular orifice sometimes occurs, and similarly interferes with the course of the circulation and the normal development of the heart; and complete transposition of the aorta and pulmonary artery has also been observed.

The ductus arteriosus and the foramen ovale may remain unclosed without any obvious reason—probably, however, from a temporary obstruction to the circulation at the time of birth; but more or less patency of the foramen ovale occurs in about 30 per cent. of healthy persons, a mere fissure or narrow valvular opening being insufficient of itself to allow of any free passage of the blood from one cavity to the other.

Instead of three sigmoid valves in the aorta or pulmonary artery there may be only two, or there may be four. This change may exist in association with other deformities, but if alone it is less likely to give rise to difficulties at birth than to lay the foundation of disease in later life.

Causation.—No more is really known of the cause of arrested or faulty development of the heart than of congenital malformations in other parts of the body.

Symptoms.—Those malformations of the heart which are dependent upon constriction of the outlet of the right ventricle (and they form a large proportion of the cases), or in any way hinder access of air to the lungs, are accompanied (of course, in proportion to their deviation from the normal) by a series of symptoms, the most prominent of which is a strongly marked lividity, due to imperfect aeration of the blood. Hence the terms *cyanosis* and *morbus cæruleus* have been used to distinguish these cases, though the former has now a more general application to all conditions of lividity, however produced. In the present case, the lividity is most marked in the prominent parts of the face—the cheeks, lips, nose, and ears, and in the fingers and toes. In slighter cases it is only richer red than natural; in the severest cases it is purple almost to blackness, and any exertion at once increases the distension of the vessels and deepens the colour. The chronic stagnation tends to cause thickening of the parts affected, and the nose and lips are coarse, while the ungual phalanges of the fingers or toes are thickened much beyond the rest of the fingers, or "clubbed." The blood shows in a remarkable degree the great excess of red corpuscles (*polycythæmia*) which is met with in many forms of cyanosis; thus the corpuscles have been found to number from 8,000,000 to 9,000,000 per cubic millimetre and the hæmoglobin may reach 110 to 160 per cent. of the normal. The patient is incapable of much exertion, from the readiness with which dyspnœa supervenes; and he is also peculiarly susceptible to cold or exposure, and easily suffers from attacks of catarrhal bronchitis. In later stages œdema of the legs, ascites, enlarged liver, and albuminuria are found; or the patient succumbs to bronchitis; or tubercular disease of the lung is the cause of death.

No one can now maintain the old view that the cyanosis of congenital heart disease is due to a mixture in the heart of venous and arterial blood. In other conditions, such as bronchiectasis and the late stages of many pulmonary diseases, great cyanosis occurs without any mixture of the two currents; and in the congenital cases probably the chief cause of the blueness is deficient aeration of the blood from obstruction of the pulmonary artery, and the circulation of the blood thus imperfectly aerated.

When, on the other hand, the pulmonary circulation is not interfered with, as in cases of simple defect in the septum ventriculorum, and in some cases of patent ductus arteriosus, the symptoms are less severe and less characteristic. Cyanosis is less marked or absent, and the symptoms are those of failure of the cardiac muscle, as in acquired valvular disease.

The **Physical Signs** do not always give precise data as to the malformation present. The præcordial region is sometimes prominent, and the dulness encroaches upon the sternum in consequence of the dilatation and hypertrophy of the right ventricle. Most commonly a systolic murmur is heard, either over the præcordial region generally; or over the base of the pulmonary artery,

since an obstruction of this vessel is so frequently a factor in the disease; or in the fourth right intercostal space, if the obstruction is at the base of the infundibulum. The murmur may be heard behind, or it is localised to the front; sometimes it is accompanied by a systolic thrill. But cases of marked cyanosis may occur without any cardiac murmur. In simple deficiency of the septum ventriculorum the blood passes from the left to the right ventricle, producing a systolic murmur, and perhaps a thrill, in the lower part of the præcordia; and a patent ductus arteriosus often causes a prolonged murmur running through systole into diastole, and waxing and waning in loudness.

Prognosis.—Congenital malformations are always unfavourable. Cases of severe defect live but a few hours or days; others of slighter degree survive five, ten, or twenty years; and even persons with very ill-developed organs have occasionally reached middle age. In any given case the prognosis must depend upon the evidences of cardiac efficiency in the history of the patients rather than upon any opinion as to the nature of the malformation. A good proportion of cases of patent ductus arteriosus live to thirty or thirty-five years of age.

Treatment.—This is entirely palliative. The patient must be kept always thoroughly warm, and protected from exposure to cold and from undue exertion. The symptoms of the later stages must be dealt with as in cases of acquired valvular disease.

HYPERTROPHY AND DILATATION

Hypertrophy is increase of size of the walls of the heart's cavities; dilatation is the unnatural distension of these cavities, and increase of their cubic capacity. The two conditions often co-exist, and hence one may have to deal with (1) simple hypertrophy, the cavity remaining of the normal size; (2) dilatation with thinning of the wall; (3) dilatation with hypertrophy.

The last occurs most frequently, hypertrophy predominating in some cases, and dilatation in others. Hence much of what is said under Hypertrophy applies to cases of hypertrophy and dilatation when the former is in excess; and much of what is said under Dilatation applies to the combined lesion where dilatation is the more pronounced.

HYPERTROPHY

Hypertrophy arises in the muscles of the heart, as it does in the muscles of the body generally, from an increased amount of work, so long as nutrition is well maintained by a proper supply of good blood. All the cavities may be enlarged together, but it will be convenient to deal with them separately.

Hypertrophy of the Left Ventricle.—The left ventricle of the heart, as having the largest amount of work in propelling the blood

through the extensive arterial system, is most often the subject of hypertrophy, and the increase of its work is most frequently due to some form of obstruction in the arterial system. The increased effort to overcome the obstruction results in hypertrophy. These causes of obstruction are: (1) Disease of the sigmoid valves narrowing the orifice; (2) constriction of the arteries, either from atheroma or very rarely from congenital narrowness; (3) degeneration of the arteries (*arterio-sclerosis*), interfering with the free circulation of the blood. A very common cause of hypertrophy of the left ventricle is (4) Bright's disease, but the exact manner in which the hypertrophy is produced is still matter for discussion (*see* Cardio-vascular Changes in Bright's Disease). (5) Excessive action of the heart from exercise, from overstrain, and from palpitation, whether purely nervous, or as a part of the disease known as exophthalmic goître, will also produce hypertrophy. Pregnancy, by the increased strain it throws upon the heart, has been credited with the production of hypertrophy, but it is at least doubtful. (6) Another cause is dilatation, which allows an abnormal quantity of blood to be present in the cavity, and hence increases the work which the walls of the cavities have to do in driving it out. (7) Mitral regurgitation is a cause of hypertrophy, by first leading to dilatation. (8) Hypertrophy may be a direct result of the action of rheumatic toxins (C. Coombs). Some doubt attaches to the view that (9) pericardial adhesions will cause it. They may be supposed to do so by opposing the contraction of the cavity and thus increasing the work of the muscle; but hypertrophy co-existing with pericardial adhesions may be really due to toxic action (8) or to dilatation following upon myocarditis.

Physical Signs.—The physical signs of hypertrophy of the left ventricle result from its enlargement and excessive action. In pronounced cases the impulse is forcible and diffused over a wide area, so as to be manifest to the eye or the hand over a space of two or three square inches by a movement of elevation communicated to the ribs as well as to the intercostal space: indeed, the chest may be permanently bulged by the enlarged heart. The impulse is often heaving, that is, slow and forcible. The position of the impulse is altered, so as to be lower and farther out than the normal: the downward displacement is pronounced in hypertrophy from aortic disease; the outward displacement is more marked under other conditions. The præcordial dulness tends to be increased in proportion to the enlargement. It may rise into the third intercostal space, may extend to the right over the right border of the sternum, and may reach externally one inch or more beyond the left nipple. It must, however, be allowed that in so much enlargement dilatation has a considerable share, and that in pure uncomplicated hypertrophy the increase of the præcordial dulness may be very slight. These physical signs are only trustworthy as long as the lungs are normal: emphysema of the lungs may completely conceal an enlarged heart; and the præcordial dulness may be increased by

retraction of a fibroid lung. The auscultatory signs are much less distinctive, as the conditions which so often cause the hypertrophy themselves modify the heart-sounds; for instance, disease of the valves, and altered conditions of the arterial circulation. Thus, valvular diseases give rise to murmurs, accompanying either the first or the second sound, as has already been explained; and in Bright's disease the cardiac sounds are habitually modified by the alterations in the arterial tension which accompany it. It is commonly said that in simple hypertrophy the first sound is less loud than normal, or muffled, and this is attributed to the thickness of muscle through which the sound from the closing auriculo-ventricular valves has to travel. The pulse, like the heart-sounds, is mostly affected by the causes of the hypertrophy rather than the hypertrophy itself, and presents the characteristics of aortic obstruction, or regurgitation, or mitral disease, or Bright's disease, or atheroma, as the case may be. In proportion to its dependence upon the heart it will be full, strong, tense, and incompressible.

Symptoms.—These are a sense of discomfort, or actual pain, about the præcordia, from the forcible beating of the heart, increased by exertion; dyspnœa on exertion; and sometimes syncope. The patient's face is said to be flushed; and headache, noises in the ears, flashes of light, mental dulness occur, as though from increased tension in the cerebral circulation. Probably hypertrophy of the heart leads to degeneration of vessels, and hence in some cases to their rupture. Where the hypertrophy results from disease of the valves, it will be only sufficient to overcome the obstruction at the orifice, and hence the arteries are not subject to undue pressure. It is different if hypertrophy arises from capillary obstruction; the arteries become in due time degenerated, and they may give way. But it is doubtful if a hypertrophied ventricle could rupture healthy arteries.

Diagnosis.—The conditions with which hypertrophy of the left ventricle is likely to be mistaken are the following: (1) Over-action of the heart from *excitement*. This condition is often seen in young people apparently well, examined perhaps for an appointment or for life insurance. The action of the heart is here generally rapid, the beat is quick and sudden, rather than slow and heaving, the patient is obviously nervous, and the condition is easily shown to be merely temporary. A murmur is sometimes heard under such circumstances (*see* p. 605). (2) The heart may be uncovered from *retraction of one lung*, usually the left; and a greater surface being in contact with the chest, it may give increased præcordial dulness, and more extended impulse. The normal position of the apex-beat, with the absence of forcible heaving, will distinguish this condition. (3) *Pericardial effusion* is frequently confounded with hypertrophy and dilatation, perhaps more often with the latter (*see* Pericarditis). (4) Displacement of the heart from new growths, or pleuritic effusion. (5) Aneurysm. (6) In all enlargements of the heart the relative share of hypertrophy and dilatation must, if possible, be

estimated. Probably no considerable enlargement takes place without dilatation. Moderate enlargements may arise acutely from dilatation. Dilatation may be inferred to be greater, the more feeble the impulse.

Prognosis.—Hypertrophy, due to causes which are not permanent, such as excessive exertion, has improved in the course of time. But with a persistent cause like valvular disease or arterio-sclerosis, no permanent recovery can be looked for, and it is much more probable that dilatation will sooner or later become a prominent feature in the case.

Treatment.—The removal of the cause must be considered. If this consists in continued overstrain, or excessive and unsuitable food, by which the arteries tend to degenerate, much may be done by rest of mind and body, by a properly regulated diet, by being careful not to overload the stomach, and by attention to the bowels; and in all cases the same principles should be followed out as a help towards improvement. With regard to drugs, it is not to be supposed that any will directly promote the diminution of the hypertrophy; but the action of the heart may be quieted when necessary by the administration of potassium bromide, or of small doses of the iodide. Digitalis is of more value where dilatation accompanies the hypertrophy, and is sometimes actually harmful where hypertrophy is largely predominant.

Hypertrophy of the Right Ventricle.—This arises, in the majority of cases, from obstruction to the pulmonary circulation, (1) at the pulmonary orifice, from congenital malformations of the valves, congenital constriction of the orifice, acquired disease of the pulmonary valves, or pressure on the base of the pulmonary artery by aortic aneurysm; (2) in the lungs, from emphysema, chronic bronchitis, bronchiectasis, and occasionally chronic phthisis; and (3) from primary disease on the left side of the heart, whereby the left auricle becomes engorged, and the pulmonary venous circulation is consequently impeded. The signs of hypertrophy of the right ventricle are analogous to those of left-side hypertrophy. A marked impulse may be seen in the epigastrium, due often to the impact of the right ventricle against the liver, rather than to direct contact of the ventricle with the abdominal walls at this point; the apex-beat may also be carried somewhat to the left. The præcordial dulness extends beyond the sternum. The pulse, if affected at all, is small on account of the difficulty the blood has in reaching the left side of the heart; or it is modified by the condition of the left ventricle. The sounds are not materially altered. Dyspnœa is present, often from the condition of the lungs.

Hypertrophy of the Auricles.—This rarely occurs without dilatation, but predominates in the left auricle in mitral stenosis (Samways). It arises from constriction or incompetence of the auriculo-ventricular valves; or from hypertrophy and dilatation of the ventricles acting back upon the auricles. Some of the forcible impulse in

mitral stenosis was attributed by Sansom to action of the hypertrophied left auricle, and variations of the mitral obstructive murmur are partly due to the same.

DILATATION

Ætiology.—The causes of dilatation of a given cavity of the heart are, on the one hand, those which tend to produce over-distension of the cavity; on the other, those which diminish the strength of the walls of the cavity, so that they yield to the force which is distending them. The cavity becomes over-filled if the blood is too quickly poured into it, as in aortic regurgitation, or if there is any obstacle to its free exit through the natural channels. In this last way nearly all the causes of hypertrophy enumerated for the different cavities of the heart are causes also of dilatation. Of these, valvular diseases, and high arterial tension in Bright's disease and arterio-sclerosis, are the most common. The yielding of the ventricular walls is promoted by various degenerative changes, some of which have been described; the fatty, fibroid, and senile degenerations, the granular or fatty conditions which follow upon anæmia, chlorosis, and severe or prolonged infections, and the myocarditis and pericarditis of acute rheumatism. The excessive indulgence in alcohol is sometimes a cause of cardiac dilatation with all its consequences. Many of these conditions are opposed to the full development of hypertrophy in the event of an obstruction to free circulation. But such predisposing causes are not essential. With considerable obstruction, the strength of the healthy muscle may be overpowered before hypertrophy has time to take place, at least in a degree sufficient to prevent dilatation.

Allied to, if not always amounting to, dilatation are the conditions of distension or at least stress, which may come on acutely or rapidly as a result of profound disturbance of the circulation; such as, over-taxation of the heart in running or other athletic exercise, or much less effort in the gouty plethoric individual, or in the patient with arterio-sclerosis. The dilatation if it occurs is generally temporary; but may be of some duration if the occasions of its occurrence are often repeated.

Anatomical Changes.—The effects on the size and shape of the heart vary with the cavity concerned. In general dilatation the heart becomes more globular, and is widened transversely. The dilated left ventricle increases to the left; and the dilated right ventricle often bulges out so as to make a prominent angle on the normally curved right border of the heart, and pushes itself in front of the left ventricle at the apex. The thickness of the walls will depend on the presence or absence of accompanying hypertrophy. In dilatation with thinning, the ventricular walls may be reduced to one-sixth of an inch, and even less at the apex, which is commonly the thinnest part. The auriculo-ventricular orifices share in the dilatation, and incompetence often results.

The consequences of dilatation are of much importance. Whereas hypertrophy is compensatory and preservative, is developed in proportion to the work the heart is called upon to do, and, in the majority of cases, does not react injuriously upon the circulation, dilatation is only the sign of the weakness of the propelling organ, and has for its effect the retardation of the flow of blood throughout the system. The dilatation of the left ventricle, with imperfect emptying of its cavity, is followed by incompetence of the mitral valve, by dilatation of the left auricle, and this in succession by engorgement of the lungs, increased tension in the pulmonary arterial system, dilatation of the right ventricle, incompetence of the right auriculo-ventricular (tricuspid) valve, dilatation of the right auricle, and congestion of the systemic venous circulation, with its secondary results—dropsy, and venous congestion of the liver, spleen, kidneys, and other organs.

Symptoms.—The general effects of dilatation are shortness of breath and palpitation on exertion, rapid irregular pulse, weakness, languor and occasional syncope; a moderate degree of anæmia, often with impaired digestion and defective nutrition. The more remote effects are seen in all the symptoms which are characteristic of uncompensated valvular disease (*see* p. 597). The immediate effects of more rapid dilatation as seen in infectious diseases have been described (*see* p. 24). The results of the sudden strain of forced athletic exercise are generally faintness going on to syncope, vomiting, and incapacity for further muscular effort; and even insensibility and death. The share which fatigue-poisons in the blood have in the causation of these last conditions is difficult to estimate.

Physical Signs.—To a much greater extent than in simple hypertrophy, the enlargement of the heart has its influence on the extent of præcordial dulness, and on the position of the impulse. The dulness is increased to the left or to the right according to the cavity mainly involved; a large portion of the heart comes into contact with the chest-walls, so that the ventricular movements are seen in two or more intercostal spaces. The impulse is carried outwards, and a little downwards. The apex-beat is generally feeble, and in some cases scarcely visible or palpable; and it is irregular, either constantly or only when additional stress is put upon the heart. The sounds in extreme cases are very faint, though clear; in others the first sound is short, clear, and somewhat high-pitched; and the second is but little affected. But just as in hypertrophy, the sounds will often depend on the valvular or arterial conditions which have helped to produce the dilatation.

Dilatation of the Left Ventricle is often accompanied by a systolic murmur, which is mostly high-pitched, of short duration, and localised to the neighbourhood of the apex. Opinions are rather in favour of this murmur being produced by vibrations in the cavity of the ventricle than of its being a murmur of mitral regurgitation. Mitral regurgitation is, however, one of the effects of dilatation, and

may be accompanied by its characteristic murmur, audible in the back. The pulse is affected by the inefficiency of the ventricle—it is small, feeble, and perhaps irregular. In extreme cases the impulse is much displaced, and may be felt even in the axilla, three or four inches to the left of the nipple. The general effects of dilatation are—shortness of breath, palpitation, and more or less cardiac distress on exertion; occasional attacks of syncope; a moderate degree of anæmia in many cases, with a not quite perfect nutrition. Digestion is also often impaired.

Dilatation of the Right Ventricle causes increase of the præcordial dulness to the right of the normal position; but also largely to the left; at the same time there may be visible pulsation in the second, third, and fourth left intercostal spaces, and in the epigastrium, often with an undulating movement, the lower spaces retracting while the upper rise. In advanced conditions it leads to a tricuspid regurgitant murmur (pp. 596, 602), and is accompanied by the indications of congestion of the systemic venous circulation, which have been described (p. 591).

Dilatation of the Left Auricle causes sometimes dysphagia from pressure on the œsophagus, and may compress the left bronchus in extreme cases.

Dilatation of the Right Auricle increases the præcordial dulness towards and beyond the right of the sternum; and, it is said, may give rise to an impulse in that situation, preceding the systole.

Diagnosis.—Dilatation has an important share in all the considerable enlargements of the heart, and it may be confounded with the several lesions mentioned under hypertrophy (p. 615). The chief physical evidence is the displacement of the impulse towards or beyond the nipple-line; and if that is produced by pleural effusion on the right side, or by aneurysm, some other proof of the existence of such a lesion will probably be afforded. The triangular dulness of pericardial effusion may be like that of a dilated right ventricle, but there are many points of distinction, which, besides the associated vascular conditions, and the history or presence of a rub, may be of value (*see* p. 633).

Prognosis.—This is unfavourable. Recent dilatation from acute disease or other temporary cause may recover; but in dilatation of any standing a return to the normal is unlikely. The best thing that can happen is the development of hypertrophy.

Treatment.—This is, in the main, the same as that of valvular diseases—rest; light but nourishing diet; the use of digitalis or strophanthus to give tone and strength to the heart-walls, combined in severe cases with direct stimulants, ammonia and brandy; and diuretics and purgatives to relieve systemic venous engorgements. In conditions of great distension of the right ventricle, venesection may be required to prevent the cessation of the heart's action. In earlier stages the general conditions, such as anæmia, and the tendency to fatty change, which have favoured dilatation, may be

dealt with by suitable means. The methods of Schott, mentioned later (*see* p. 628), may be useful in some of these severe cases.

In acute cases, brandy, ether and strychnia, internally or by subcutaneous injection, are necessary; followed by a considerable period of rest.

MYOCARDITIS

Myocarditis, or inflammation of the muscle of the heart, may be either acute or chronic. *Chronic myocarditis* can only be recognised in its final stage of fibroid change, and is included in the description of fibroid degeneration (*see* p. 623).

Acute myocarditis occurs mostly in connection with pericarditis or endocarditis as a part of rheumatic fever. In fatal cases of pericarditis, the layer of muscular tissue immediately under the pericardium is sometimes seen to be paler than normal, as if fatty; and in cases of adherent pericardium, bands of fibrous tissue may be found extending from the surface into the substance of the heart. According to Dr. C. Coombs rheumatic myocarditis in young persons shows the following changes: In the muscular fibre-cells fatty granules, and droplets first appearing near the nucleus; in the interstitial tissue, first, nodules consisting of large fusiform cells, of which many are multinuclear, and secondly, collections of leucocytes, chiefly polymorphonuclear. The nodules are closely related to the arteries and arterioles, occur deeply in the muscular wall and near the root of the aorta and the mitral ring. They are more common in the left ventricle, and they terminate in cloudy swelling or fibrous transformation (cicatrix).

A more local inflammation of the heart's muscle results from malignant endocarditis, where ulceration of a valve extends to its base, and then invades the muscle; or where vegetations or semi-detached fragments set up ulceration in adjacent parts of the endocardium by friction or contact, and this involves the myocardium. The effects upon the general circulation may be of the same kind as those of malignant endocarditis.

A third form is *suppurative myocarditis*, which is chiefly the result of pyæmia. Small abscesses occur in the substance of the heart, mostly in the wall of the left ventricle, and may approach so near to the pericardium as to rupture into its cavity and set up acute pericarditis. This form of myocarditis occurs especially in connection with, and secondary to, acute necrosis of the long bones (*see* Pyæmia, p. 162).

The **Symptoms** of myocarditis are by no means pronounced. It diminishes the contractile power of the heart, the first sound is fainter, and the pulse becomes feeble or irregular; there may be dyspnœa and tendency to collapse; but its share in producing these when pericarditis or endocarditis is present will be difficult to estimate. Dilatation with increased præcordial dulness is also frequent; but it is open to doubt whether this is caused by the fact

of myocarditis, or by the influence of the toxins which are in operation. A similar uncertainty attends the systolic mitral murmur which is often present: it may be due to yielding of the mitral ring, or to co-existing endocarditis.

The symptoms of abscess of the heart are also uncertain, and the diagnosis from physical signs is equally obscure.

Prognosis and **Treatment** have little place where diagnosis is so untrustworthy; but the form associated with rheumatic pericarditis is the only one that can be looked upon as curable, and here the treatment pursued for the relief of the accompanying lesion will be applicable; the support of the failing heart being the main indication.

DEGENERATION OF THE MYOCARDIUM

The muscular wall of the heart is liable to the following forms of degeneration—*pigmentary*, *fatty*, and *fibroid*.

PIGMENTARY DEGENERATION

(*Brown Atrophy of the Heart*)

The heart is smaller than normal, and the muscular fibre, instead of having a full red colour, is of a dull brownish red, and softer and more friable than is natural. Under the microscope the fibrillæ are seen to contain a number of minute yellow granules. It occurs in senile and cachectic conditions, being common in fatal cases of malignant disease of other organs.

FATTY DEGENERATION

This change in the muscular fibres must be distinguished from the deposit of fat about the heart (*see* p. 627). In the latter the ordinary adipose tissue is deposited beneath the pericardium, and invades the muscular fibre by the growth of fat-cells between and amongst them. In the former, or true fatty degeneration, the muscular fibrillæ themselves are the seat of minute fat granules, which replace the true sarcous elements and rob the muscle of so much of its contractile tissue. This true fatty degeneration occurs in different forms; the muscular wall may be uniformly affected, or the fatty changes may be limited to a small patch, or to the layer underlying the pericardium, as described under Myocarditis, or it may consist of streaks and lines on the inner surface of the heart. When the affection is general the heart is of softer consistence, more easily lacerable, of pale pink or buff colour, and often somewhat larger than normal, from yielding of the affected muscular tissue. When the fat is deposited in lines or streaks it gives a characteristic appearance, the lines of pale yellow colour being

often arranged upon the darker red muscle, like the markings of a tabby cat. They are seen mostly on the musculi papillares, on the posterior wall of either ventricle, and on the septum in the right ventricle. Fatty degeneration is common in hypertrophied hearts; and may be present even when the muscle has a quite normal colour.

Ætiology.—The causes of fatty degeneration of the heart are general and local. It may be the result of a general tendency to degeneration, such as occurs at an advanced age; it is seen constantly in pernicious anæmia, and often in other forms of anæmia, in purpura and scurvy, and in cachectic conditions, such as phthisis and cancer; in poisoning by phosphorus, by some mineral substances (lead, antimony, arsenic), and in chronic alcoholism. In most of the acute febrile diseases the consistency of the heart is sometimes altered, as the result of a finely granular condition of the muscular fibres, which is probably not to be separated from fatty degeneration. This is the case in enteric and typhus fevers, in yellow fever, diphtheria, small-pox, and measles. The most important local condition is obstruction or narrowing of the coronary artery, by which the nutrition of the heart-wall is necessarily impaired. This may arise from atheromatous or syphilitic changes in the vessels, or from thrombosis or embolism. Fatty degeneration also arises from myocarditis, and is seen in connection with long-standing valvular disease, with renal disease, and in hypertrophy or dilatation from other causes.

Symptoms.—In many cases the fatty change does not materially affect the symptoms due to the associated pathological condition of the heart. The physical symptoms attributed to the more pronounced fatty conditions are—feeble cardiac impulse; faintness of the cardiac sounds, with murmurs if there is dilatation; a slow, feeble, and sometimes irregular pulse. The chief symptoms are pallor, attacks of syncope, and dyspnœa, chiefly on exertion, but sometimes constant. The syncopal attacks may be nothing more than transient faintness, or there may be complete unconsciousness. Occasionally they have much resemblance to cerebral attacks, with a sudden fall, coma, stertor, and convulsive twitchings; or they may be like epileptic fits. Recovery from the attacks takes place without hemiplegia. The association of these cerebral symptoms with a pulse of less than forty constitutes Adams-Stokes disease (*see* p. 624). Sometimes the dyspnœa has the characters of Cheyne-Stokes respiration. Slight œdema of the feet may be present, but rarely a well-marked dropsy, and there is almost entire absence of the signs of venous congestion which are seen in valvular disease. In other respects the functions of the body are badly performed, and nutrition is imperfect. The tissues are soft, muscular power is diminished, the appetite is poor, and digestion is bad. Death may happen in one of the syncopal attacks, or quite suddenly, or by a more or less prolonged asthenia. In a certain proportion of cases, rupture of the heart takes place. In the course of severe pyrexial illnesses, like

typhoid fever, the following symptoms suggest the occurrence of granular or fatty change in the myocardium. The pulse, hitherto rapid, becomes weak and irregular, the cardiac impulse is feeble, the apex-beat is displaced outwards, and the first sound is so faint as to be scarcely audible. The patient becomes pale and in later stages livid.

Diagnosis.—It is obvious that fatty degeneration does not always give sufficiently clear indications for its recognition. The evidences of feeble cardiac action, apparently not due to valvular disease, or to ordinary dilatation, together with marked dyspnœa, or the peculiar syncopal attacks in a person of advanced age, are the chief points to note. Degenerative signs elsewhere are of little value, but their absence might be regarded as opposed to the diagnosis.

Prognosis.—Except in the form that occurs in acute illnesses, the condition is probably incurable.

Treatment.—This must consist in avoidance of undue exertion, of making efforts with the breath held, or of mental excitement; in the use of a diet, with more nitrogenous food and less of the fatty, starchy, and saccharine elements; in moderate doses of stimulants, and the exhibition of tonics, such as quinine, arsenic, iron, and strychnine. Digitalis must be given with caution, and only in cases where the beat is frequent and irregular, with evidence of dilatation.

Cardiac failure in enteric fever requires the free employment of stimulants, with ammonia and digitalis in frequent doses.

FIBROID DEGENERATION

In this form of degeneration, the muscular tissue of the heart is replaced by white fibrous or connective tissue. The change is in most instances partial, so that streaks and patches of a white, yellowish-white, or gray colour are seen deep in the muscular substance. It affects the lower third of the ventricle, the lower third of the septum, the musculi papillares, and sometimes the bases of diseased valves. Only occasionally is the ventricle almost entirely converted into fibrous tissue, but even here some traces of muscular fibre may be found on microscopical examination. A very slight degree of this must often result from rheumatic myocarditis; and in more pronounced cases the co-existence of pericardial or endocardial lesions will sometimes show its inflammatory origin (*chronic myocarditis, interstitial myocarditis*), but it is often degenerative and is consequent upon malnutrition from obstruction of the coronary arteries. It has been ascribed also to alcoholism, to long-continued congestion, and to syphilis (*syphilitic myocarditis*). The patch of fibrosis may be remote from the vessels, a secondary result of infarct (*para-arterial*); or in the immediate neighbourhood of the vessel (*peri-arterial*) by extension from its outer wall (Cowan). The heart affected with fibroid disease is generally hypertrophied, and

it may be dilated, or the subject of adherent pericardium; the affected part of the heart's wall is often thinner than normal, and it may be bulged out into a distinct aneurysm.

The **Symptoms** of fibroid degeneration are not distinctive. In some cases, apparently perfect health has been enjoyed till the patient has suddenly fallen dead; in others, the symptoms have been those of valvular disease. There may be in these instances evidence of dilatation, or the murmur of mitral regurgitation.

The **Treatment** is that of cardiac dilatation or valvular disease.

ADAMS-STOKES DISEASE

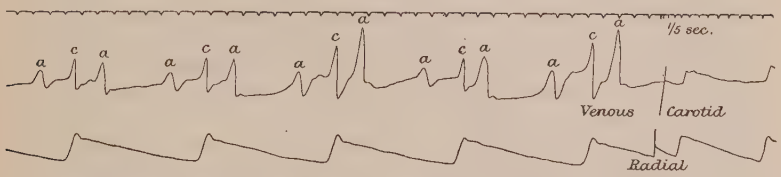
The clinical condition to which this name has been given may be here described, because its chief organic cause is disease of that bundle of muscular fibres in the interior of the heart, which is known as the auriculo-ventricular bundle of His, and which has already been described (*see* p. 577).

It was observed by R. Adams in 1827, and by W. Stokes in 1846, that patients with abnormally slow pulse might be subject to attacks of syncope, unconsciousness, or convulsions; and the pathological basis of such cases has now been ascertained as a result of our knowledge of the anatomy and physiology of the bundle of His, and of the clinical observation that in such cases many auricular beats fail to reach the ventricle. Stokes, indeed, observed that the veins in the neck were beating faster than the radial pulse: and in two of these earlier cases there was fatty degeneration of the heart, in which the bundle of His must have been involved.

Symptoms.—The patient is generally one somewhat advanced in years; he may, indeed, be quite old when he first suffers from an attack of faintness, or syncope, often leading to unconsciousness. The face is pale, or ashen grey or livid, and may flush later; the pupils generally dilate; the breathing may cease for a few seconds, and when resumed it is stertorous. With this there may be twitching of the fingers or of the facial muscles. Sometimes there are more marked convulsions, but not often the extensive general convulsions of epilepsy. But there is great variety in the attacks; they are described as syncopal, apoplectiform or epileptiform; and there is sometimes a definite aura. When, on the first attack, the pulse is felt, it is found to be abnormally slow, as infrequent as 30, 20, or even 6. The patient in a short time—three, five, or ten minutes—recovers his consciousness, and the pulse may return to its normal frequency. The attacks recur at irregular intervals of days, weeks, or months; with each recurrence the pulse again becomes slow, and as the case proceeds the infrequency of the radial pulse becomes persistent in the intervals as well as in the attacks. The examination of the heart may be negative; in some cases, however, aortic disease is shown by characteristic murmurs, in others there is evidence of dilatation, and in others

a systolic apex murmur with each radial beat. In many cases auscultation will detect cardiac sounds occurring alternately with those which correspond to the radial pulses; and these are in all probability due to auricular contractions. If the jugular vein be examined, it will be found to pulsate twice as frequently, or three

FIG. 63



A Polygraphic Curve from a Case of Heart-block. The upper tracing is from the veins of the neck, the lower tracing from the radial artery. Each radial beat is accompanied by a systolic elevation, *c*. At regular intervals in the curve additional waves due to auricular systole are present, marked *a*. The auricle is contracting twice as frequently as the ventricle. (*After Lewis.*)

FIG. 64

An Electro-cardiogram taken with the String-galvanometer from a Patient with Adams-Stokes Disease. The rhythms of auricle and ventricle are dissociated, and the ventricle is no longer responding to the auricle. The ventricular systole produces the displacements R and T; the auricular systoles produce the movements P, which are distributed uniformly through the tracing, and have no constant relations to the ventricular movements; but they are nearly twice as frequent. (*After Lewis.*)

or four times as frequently, as the radial pulse; and tracings taken from the jugular vein and radial pulse simultaneously with the polygraph will demonstrate this (*see* Fig. 63). A similar condition is shown by the electro-cardiogram (*see* Fig. 64).

This disturbance of function, in which the contractions of the auricle are not uniformly and constantly, or even not at all, conducted to the ventricle, has already been described as *heart-block* (*see* p. 562); and it has now been invariably found in cases presenting the symptoms of Adams-Stokes disease.

In most cases the heart-block is partial, but the symptoms have also been observed with complete dissociation of the auricular from the ventricular beats, or *complete* heart-block.

Cases of this disease usually follow a downhill course: the fits become more numerous, the pulse is persistently slow, and death takes place in one of the apoplectiform attacks; or the patient

lapses into semicoma, or coma with perhaps Cheyne-Stokes breathing, and the heart finally fails completely. But occasionally the symptoms have subsided.

Morbid Anatomy.—Since the cases have been properly understood, a lesion of the heart involving the auriculo-ventricular bundle has been constantly found, in the form of extensive fatty degeneration, or of fibrous changes extending perhaps from the base of the aortic valves, or of syphilitic gummatous tissue or tumour. The explanation of the symptoms is the opposition by the diseased bundle of His to the transmission of auricular impulses to the ventricles, and the interference with the cerebral circulation, by the defective contractions of the ventricle.

Treatment.—In view of the pathology of the disease, little can be done, unless it should be suspected that gummata are present, when potassium iodide and mercury should be given. Otherwise the treatment must be that suitable to the apparent cardiac lesion, or to arterio-sclerosis.

RUPTURE OF THE HEART

Apart from injury, this is mostly a consequence of fatty degeneration ; in a very small proportion of cases, its cause is abscess, malignant endocarditis, or aneurysm. It occurs, like fatty degeneration, in old people, and not infrequently follows muscular efforts. The left ventricle has been the seat of the rupture in three-fourths of the cases on record. The patient is suddenly seized with intense cardiac pain, followed quickly by pallor, unconsciousness, a few convulsive twitchings, and death. In rare cases life has lasted some hours, or even days, with pallor, cold sweats, feeble pulse, and sighing respiration. It may then closely resemble the rupture of an aneurysm into the pericardium. Absolute rest, with the head low, maintenance of bodily warmth by external applications, and of the circulation by small quantities of stimulants frequently administered, are clearly the indications for the almost hopeless treatment of such a condition.

ANEURYSM OF THE HEART

Aneurysms of the heart may be acute or chronic.

Acute aneurysms arise from ulcerative endocarditis of the ventricle wall, in the manner mentioned under Endocarditis, and this is a rather frequent cause of aneurysm of the pars membranacea septi, as well as of the valves (*see* p. 582). Aneurysms of the undefended space are sometimes congenital. In either case, the sac opens towards the left ventricle. The condition is not recognisable during life.

Chronic aneurysms of the heart commonly arise in connection

with fibroid degeneration. The cavity affected is weakened at one spot by this conversion of its muscular fibre into fibrous tissue, and dilates under the pressure of the blood into a sac. The left ventricle is their usual seat, and only a few cases are on record of aneurysms of the other three cavities. In two cases out of three they occupy the apex; they form rounded sacs, of which the communication from the ventricle may be of the same size as the sac itself, or very much smaller. The former variety is more frequent when they arise at the apex—that is, the aneurysm is continuous with the cavity of the ventricle; the latter, more sacculated variety, occurs more often at the side or the base of the ventricle. In size they have been compared to nuts, fowls' eggs, or small oranges; a few have been much larger. The walls are generally very thin, and sometimes infiltrated with calcareous matter; they are lined by endocardium, and mostly contain fibrinous coagulum. They have been found at all ages, from twelve upwards; and in males more often than in females.

Symptoms, if present, cannot be distinguished from those of other cardiac and valvular lesions, with which the aneurysm may indeed be associated; but in a considerable proportion of cases death has taken place either suddenly, and probably from syncope, or, as afterwards proved, from rupture of the aneurysm into the pericardium.

FATTY OVER-GROWTH

In this condition, which must be distinguished from fatty degeneration, the surface of the heart is overlaid by a large quantity of fat, so that the muscular fibres may be entirely concealed from view. The fat, which is simply an over-growth of adipose tissue beneath the pericardium, encroaches upon the muscular wall, and, pressing upon the muscular fibres, causes them to waste to a certain extent. It occurs for the most part in persons suffering from general excess of fat, or obesity.

Symptoms.—In the more pronounced cases—like those of fatty degeneration proper, the result of enfeebled action of the heart—there are diminished impulse, faint sounds, and weak, small pulse; dyspnœa on exertion, and occasionally attacks of syncope. Death takes place suddenly in some instances. The symptoms may be in part due to accompanying atheroma of the arteries.

Treatment.—The treatment of fatty infiltration of the heart should be conducted much on the same lines as that of *obesity*, with due consideration of the fact that the heart is affected. Excess in eating and drinking should be avoided; alcoholic beverages should be forbidden. The necessary fluids are better taken shortly before or two hours after a meal than with it. The diet should consist chiefly of lean meat, chicken, fish, green vegetables, and ripe fruit, while starchy, saccharine, and fatty foods should be reduced to a minimum.

The system of treatment conducted by Schott and others at Nauheim is probably more suitable to fatty over-growth of the heart than it is to severe valvular diseases.

It consists partly of immersion in saline baths, partly of regulated exercises of the muscles of the arms, trunk, and legs conducted slowly against resistance on the part of an attendant. The different springs of Nauheim have a temperature of from 60° F. to 95° F., and contain free carbon dioxide, in addition to the salines, of which the most abundant are sodium chloride, and calcium chloride and bicarbonate. An important influence is attributed to the stimulating action upon the skin of the saline ingredients, and the minute particles of carbon dioxide gas. An elaborate series of movements has been devised, comprising flexion and extension at all the large joints, these movements being made by the patient, but gently opposed throughout by the attendant. No movement is repeated at the same sitting, an interval of rest occurs between any two successive movements, and indications of strain or dyspnœa or distress are the signals for ceasing the exercises. The results of this combined treatment are stated to be slowing and lessened irregularity of the pulse, great diminution of the præcordial dulness, and general improvement in the comfort of the patient.

It is allowed that the waters of Nauheim may be successfully imitated by the addition to other waters of equivalent proportions of sodium chloride (1 to 3 per cent.) or calcium chloride (·2 to ·5 per cent.), or sufficient quantities of bicarbonate of sodium and hydrochloric acid to cause effervescence.

Oertel's method combined systematic hill-climbing with a diet in which fluids and fat-forming elements were reduced to a minimum; but it cannot be recommended.

NEW GROWTHS AND PARASITES

Under this head we may shortly mention *tubercle, cancer, syphilis, hydatids,* and *cysticerci.*

Tubercle.—Tubercles not infrequently form in connection with inflammation of the pericardium (*see* p. 631), when they are found as whitish-gray or yellowish granulations, mostly in the substance of the pericardial lymph or false membrane uniting the layers of the cavity, or sometimes actually under the layer of the visceral pericardium. They occur in the course of general tuberculosis, or at least secondarily to tubercle of the bronchial or mediastinal glands, or of the lungs and pleura. The diagnosis can only be made from the appearance of pericarditis under those circumstances; but it must be remembered that a pericarditis, which is not tubercular, may also arise in the course of phthisis. Isolated deposits of tubercle are exceedingly rare.

Cancer.—This attacks the heart in different forms, chiefly as lymphoma, sarcoma, and melanotic cancer. Frequently the heart

is affected as a consequence of sarcoma or lymphoma of the mediastinal glands; the tumour then spreads along the veins, invades the auricles, and appears as nodular elevations under the pericardium. Sometimes the tumour is secondary to a similar growth in another part of the body. A primary isolated deposit in the heart is exceedingly rare. There are no symptoms that are distinctive of cancer of the heart. It could only be inferred, in certain cases, from the existence of intra-thoracic growth. Walshe records a case where the conversion of the anterior wall of the right auricle into cancer was unattended with any clinical evidence pointing to the heart.

Syphilis.—The lesions of this disease occur as arteritis, as fibrous scars (*syphilitic myocarditis*), as fibroid masses, or as distinct gummata, which may be cheesy, and even softening in the centre, affecting the muscular substance of the heart in the same way as the voluntary muscles and causing some surrounding inflammation. The gummata are seated chiefly in the walls of the ventricles. They produce no characteristic symptoms, but may be the cause of irregularity, attacks of angina, or syncope, with evidence of enlargement if the myocarditis is extensive. If the lesion is in the septum of the ventricles, it may cause the characteristic symptoms of Adams-Stokes disease. Sudden death has rather frequently occurred.

Parasites.—*Hydatids* occasionally develop in the substance of the heart, forming cysts which project in the course of their growth either towards the pericardium, or into one of the cavities. The cysts are single, or may contain daughter-vesicles. Their effect upon the heart depends, of course, upon the size to which they grow. They have been found *post mortem* in death from causes independent of the heart; but in many cases they have been directly fatal, either by escaping from the wall of the heart into its cavity, or by rupturing and discharging daughter-cysts into the interior or into the pericardium. In the former case the daughter-cysts may get impacted in branches of the pulmonary artery, and cause death rapidly; or in a large artery of one of the limbs, and thus bring about gangrene. A case is recorded of rupture at the same time into the ventricle and into the pericardial cavity, so that hæmo-pericardium resulted. Naturally the existence of a considerable cyst would interfere with the action of the heart or of its valves, but there is nothing by which such disturbance could be referred during life to hydatid, unless it could be shown by the Röntgen rays, or a cyst were known to exist elsewhere, or a serum reaction could be obtained.

The *cysticercus* of the tænia solium is also sometimes found in the walls of the heart.

PERICARDITIS

Ætiology.—Inflammation of the pericardium may result from a general blood-poisoning; or it may occur from direct irritation or infection of the serous sac.

Among the first class of cases, acute rheumatism is its most frequent cause; it occurs in Bright's disease, in pyæmia, in leuchæmia, in tuberculosis, in influenza, in general pneumococcal infection, and in other conditions of septicæmia and toxæmia. Its local causes are the growth of cancer nodules into its cavity, the rupture into it of abscesses and hydatid cysts, and the contiguity of a source of infection, such as empyema or pneumonia.

Anatomical Changes.—If we take as the type the pericarditis which occurs in the course of acute rheumatism, we find the following changes: In early stages the membrane loses its smooth, glossy surface, and becomes more vascular, so that it is injected with a fine network of vessels. Some shreds of lymph from the exudation of corpuscular elements and fibrin from the blood-vessels are next seen, and a complete layer forms upon the pericardium. Ultimately the two opposed surfaces of the sac may be separated by a layer of lymph one-eighth or a quarter of an inch in thickness, which is sufficiently soft to allow the parietal and visceral membranes to be peeled from one another, and the lymph is often of such a consistence that the separation of the surfaces leaves a curiously honeycombed or reticulate appearance. Generally, at the same time, some serum is formed, of yellow colour, and turbid from corpuscular elements. This may accumulate to a considerable amount and further separate the two layers of the pericardium, while it allows the formation of long shaggy processes of lymph, stretching from surface to surface. After a time the fluid generally disappears, and the lymph is either itself absorbed, or it becomes organised, and unites the parietal and visceral layers of the sac more or less completely together. In this process new vessels grow in the investing lymph, and fibres of connective tissue are gradually developed. The amount of firm connective tissue thus formed and the completeness of the union effected vary much in different cases; there may be a few fibrous bands crossing the cavity, or a dense layer of tissue half an inch thick. The mediastinal connective tissue is sometimes involved in inflammation at the same time, forming *mediastino-pericarditis* or *mediastinitis fibrosa* (*see* p. 664. *See also* Polyorromenitis).

Variations in this process take place. Under certain circumstances, mostly in pyæmia or septicæmia, the fluid contents of the pericardium are pus, instead of serum, constituting *purulent* or *suppurative pericarditis*. This is often secondary to abscess of the cardiac muscle, which is known frequently to result from acute necrosis of the long bones. Sometimes the new-formed vessels in the inflammatory formation rupture, and small petechiæ or larger patches of

hæmorrhage cover the surface of the membrane, forming *hæmorrhagic pericarditis.* And, occasionally, tubercles are formed both in the new tissue and in the original membrane covering the heart's surface; this is known as *tubercular pericarditis,* and forms part of a general tuberculosis.

The micro-organisms of pericarditis vary with its origin. Streptococci, staphylococci, pneumococci, and tubercle-bacilli have been most often found. Poynton and Paine found their rheumatic diplococci in the pericarditis of rheumatism.

Physical Signs.—Since pericarditis so frequently arises in the course of some infectious disease, like rheumatism, its symptoms may be entirely masked by those of the disease which it accompanies, and its presence may be only revealed by the alteration in the heart-sounds, and other physical sounds which it produces. These, however, are generally characteristic. In the early stages a slight shuffling sound is heard in addition to, though partly obscuring, the normal sounds. The shuffling consists of two sounds occurring during systole and diastole respectively, but not always absolutely synchronous with the first and second sounds; it is heard over the præcordial region, often first at the base, later over the whole of the area corresponding to the anterior surface of the heart and pericardium. Frequently this *pericardial rub* has a triple character, like the pace of a cantering horse. After a short time the sound becomes louder and harsher, resembling the friction of hard rough surfaces upon one another, and when it has reached this stage the friction can often be felt by the hand placed over the præcordial region.

If liquid is effused into the pericardium, as is frequently the case, the præcordial dulness is increased. It extends upwards to the upper border of the third rib, the upper border of the second rib, or even to the clavicle; towards the right for one inch or more over the sternum; and towards the left for an inch or so beyond the left nipple. The præcordial dulness has a more or less triangular shape, with its broad base upon the diaphragm, and a rounded apex at the upper part of the sternum and the left upper intercostal spaces. In this extension of præcordial dulness, the small area of resonance in the fifth right intercostal space normally corresponding to the sharp angle between the convex border of the right auricle, and the upper surface of the diaphragm, becomes obliterated. This is called *Rotch's sign;* but its diagnostic importance is somewhat doubtful.

As the fluid increases, the impulse of the heart moves gradually outwards and upwards, until with much effusion it may be felt in the third left space above and external to the nipple; this is not because the apex is raised, but because the impulse is formed by a part of the heart more and more remote from the apex. An important distinction must here be mentioned between pericarditis and pleurisy in the effect of effusion of fluid on the occurrence of a friction sound. In pleurisy the effusion of fluid results in the disappearance of the pleuritic friction sound. In pericarditis, the friction

sound persists commonly throughout the illness, even to the period of greatest distension of the sac, and during the subsequent absorption of the fluid. In the case of the pleura the effusion compresses the lung and separates the two pleural surfaces from one another; in the case of the pericardium, the effusion finds less resistance on the side of the lungs, and thus increases præcordial dulness, while between the solid heart and the spine and sternum, behind and in front, there is but little room for the accumulation of a large quantity of liquid. According to Ewart, pericardial effusion causes a dull area at the base of the chest behind, corresponding to the eleventh and twelfth dorsal spines, quadrilateral in shape, measuring about four inches vertically, and from five to six inches across, of which about two-thirds are to the left of the middle line, and the remainder to the right: this he attributes to damping of the percussion-note over the liver by the pericardial bag of fluid above it. Apart from his, if the effusion is extensive the left lung is compressed, and dulness, bronchial breathing, and diminished tactile vibration occur at the left base below the angle of the scapula (*see* p. 505).

Another result of pericarditis in some cases is inhibition of the action of the diaphragm. Either abdominal respiratory movements cease, or there is a little recession during inspiration, with upward movement of the higher abdominal viscera, and of the heart, collapse of the bases of the lungs, and it may be distension of the stomach and colon.

Symptoms.—The local symptoms which may accompany pericarditis and pericardial effusion are pain, anxiety or distress at the præcordia, tenderness on pressure over that region, shortness of breath, with shallow respirations, and short hacking cough. The pulse may not at first be much affected, but it tends soon to be faster and fuller, and in the later stages of effusion to become feebler, and even fluttering and irregular. Occurring in the course of a febrile disease like rheumatism, it may not notably add to the existing pyrexia, but with its rapid onset occasionally there is considerable elevation of temperature—for instance, to 105° or 106°; and in other cases it tends to produce the usual conditions of pyrexia, loss of appetite, dry tongue, thirst, and scanty urine.

In the worst cases the cardiac feebleness increases, the pulse becomes irregular and fluttering, præcordial anxiety is severe, and the face becomes drawn and pinched; and the dyspnœa is no doubt aggravated by the pressure of the distended pericardial sac upon the left lung. Ultimately, the nervous system fails, and delirium, jactitation, convulsion, or coma ends the scene. But in the majority of instances the symptoms gradually subside; the dulness diminishes from above downwards, while the rub often remains till a late stage. In many cases, no doubt, adhesion of more or less of the pericardial surface takes place.

The changes of pericarditis occur rapidly, effusion may reach its height in two or three days, and subsidence may be well established in three or four more.

Suppurative, tubercular, and hæmorrhagic pericarditis are not essentially different in their symptoms and physical signs.

Diagnosis.—Under ordinary circumstances this presents no difficulties, the double or triple friction sound being very distinctive. A double pericardial rub may, however, sometimes be simulated by a *double aortic murmur,* and if there is simultaneous dilatation of the right ventricle, the outline of pericardial effusion may be closely imitated. The co-existence of rheumatic fever will not always help, as it might accompany both pericarditis and old aortic disease. The pericardial friction sounds are, however, less likely to be limited to the area of aortic valve murmurs; they are often not strictly synchronous with the heart-sounds, are not accentuated at the commencement, are often increased by pressure with the stethoscope, and vary within a few hours in their relative intensity at different parts of the præcordia. The diagnosis of pericardial effusion is not always easy, because it may be simulated by a *dilated heart,* caused by the same rheumatic poison as has led to the pericarditis. The signs which support the diagnosis of effusion are Rotch's sign, a straight right border of the dull area as compared with the convex outline of the right auricle, extension of dulness to the left beyond the heart's impulse, elevation of the impulse to the fourth or third space, signs of compression at the left base, and marked systolic retraction of the epigastrium. The Röntgen rays show that the space between the apex of the heart and the diaphragm is obliterated, and with a large effusion the heart's shadow may be seen within a ring of shadow due to the distended pericardium.

Prognosis.—Pericarditis is not, on the whole, an immediately fatal disease. It may be so mild that it is only detected by the stethoscope in the course of a routine examination, and in a large proportion of the cases occurring in rheumatic fever the inflammation subsides. The adhesion of the layers, which so often results, may become a danger in itself. In Bright's disease, and in association with other chronic cachexiæ, it often occurs towards the end of the illness, and then may appear to be the lesion determining death; but even in such circumstances the physical signs may completely disappear before death, or, if they persist, the fatal result may not seem to be hastened thereby. The recognition of the associations suggestive of tubercular or purulent pericarditis will make the prognosis a grave one, and the occurrence of a pneumococcal pericarditis in the course of pneumonia or empyema is generally fatal: but I have known a case of double empyema with pericarditis recover. Pericarditis in acute rheumatism is often accompanied by, and masks, some inflammation both of the endocardium and of the myocardium, the ill-effects of which become developed afterwards.

Treatment.—The treatment of pericarditis is mainly palliative. Like other acute inflammations, it must be met by complete rest in the recumbent or semi-recumbent posture, by nutritious fluid diet, and by abstinence from talking, excitement, or worry. In the

case of rheumatic fever, these conditions are probably already provided in the treatment of the initial disease, which may be continued, provided that the heart's action is not seriously weakened by any drug—*e.g.*, salicylates in excess. The further indications required by the implication of the heart resolve themselves into the relief of pain, the maintenance of the strength of the heart and circulation, and the absorption of effused fluid when this subsides slowly. For very severe pain six or eight leeches may be applied to the præcordia; but the same effect may be obtained by the use of morphia internally, or by subcutaneous injection. The præcordia may be protected by a layer of cotton wool, or a warm linseed-meal poultice may be applied. If the circulation tends to fail, or the heart becomes irregular, small doses of tincture of digitalis should be given frequently, with brandy or ammonia. Medicinal measures for the removal of fluid in pericarditis are uncertain. As a rule, it soon begins to be absorbed if the heart can be sustained during the short period when it is at its height. If a considerable quantity remains for some time, iodide of potassium may be given. If the liquid is in excessive amount and threatens a fatal result, or if it is presumed to be purulent, it may be removed by operation. An incision is generally made over the fifth left interspace, about one inch from the sternum, where the pericardium is exposed and can be aspirated: but the fifth or sixth interspace outside the nipple line is probably preferable. If the liquid is purulent, a free opening may be made by removing the fifth costal cartilage, and the sac can be washed out and drained.

ADHERENT PERICARDIUM

Reference has been already made to this condition as arising from pericarditis. The degree to which the two surfaces may adhere varies much in different cases; there may be merely a few filaments running from the surface of the heart to the parietal pericardium, or there may be complete union of the pericardial sac to the surface of the organ, and every intermediate condition occurs. When the union is complete, the tissue uniting the two surfaces may form only a thin layer; or it is a dense, firm, fibrous, more or less vascular coat, a quarter or even half an inch in thickness. In rare cases, also, calcareous matter is deposited in the adhesions, so that a complete investment by it may take place. Although the serous sac of the pericardium seems especially devised to allow the free movement of the heart, the simple adhesion of the two layers is not necessarily followed by any ill-effects upon the form and size of the heart, and in a certain proportion of cases the heart has its normal size. But in others hypertrophy or dilatation is present. In many of these last valvular disease is associated with the pericarditis, and sufficiently accounts for the changes in the heart-walls. If the pericardial adhesion is very

extensive and dense, dilatation and hypertrophy may occur without being accounted for by any valvular disease, and it is here probable that the muscular substance of the ventricle has been injured by the occurrence of myocarditis at the same time as the pericarditis. In some cases there is not only obliteration of the pericardial sac, but the external surface is firmly fixed to the surrounding pleura and to the sternum, and the adjacent pleural layers are also adherent (*mediastinitis*).

Symptoms and Physical Signs.—The symptoms associated with adhesion of the heart to its pericardial sac are for the most part, if not entirely, due to dilatation and hypertrophy of the cardiac walls: anginal pain, palpitation, and dyspnœa may be especially mentioned. Physical signs cannot be relied upon to reveal the presence of this adhesion itself; it has been often found *post mortem* when unsuspected; and it may only be inferred from the knowledge that an acute pericarditis has previously occurred. But when the more extensive external adhesions are also present, one or more of the following physical signs may be recognised: (1) systolic retraction at the point corresponding to the apex of the heart; (2) systolic retraction of the lower end of the sternum; (3) systolic retraction of the third, fourth, and fifth intercostal spaces to the left of the sternum; (4) systolic retraction of the lower ribs at the side or back of the left chest (J. Broadbent); (5) a diastolic rebound or shock after the systolic retraction at the apex; (6) absence of alteration in the præcordial dulness, and in the position and force of the impulse during respiratory movements; (7) sudden collapse of the veins of the neck during ventricular diastole (Friedreich); (8) failure of the sternum to advance during inspiration (Wenckebach). These are not all pathognomic; certainly systolic retraction of intercostal spaces is not peculiar to adherent pericardium, much less is systolic recession of the epigastrium. Some of the others are difficult to verify in particular cases, and others are not constantly present. Since adherent pericardium so often occurs in company with valvular and myocardial lesions, the physical signs, as well as the symptoms, of the latter are apt to be credited to the former. But a systolic, and sometimes even a presystolic murmur may occur without valvular disease when the heart is dilated as a result of adherent pericardium. Adhesions may sometimes be inferred, when the patient is suffering from evidence of failure of the right ventricle, such as dyspnœa, dropsy, enlargement of the liver, and albuminuria, without any obvious cause for the right-sided failure, such as mitral or pulmonary disease. This diagnosis is still more reasonable if there is or has been pleurisy or pleuritic effusion or adhesions on one or both sides, as this increases the probability of a combined pleural, pericardial, and mediastinal inflammation (*mediastinitis*) having been present.

The **Prognosis** and **Treatment** of pericardial adhesions must be considered chiefly in reference to the changes in the structure and functions of the heart which result from them (*see* Hypertrophy

and Dilatation). In exceptional cases, with pronounced evidence of external adhesions, other measures may be taken (*see* Mediastinitis).

PERICARDIAL EFFUSIONS

HYDROPERICARDIUM

This term is intended to denote the presence of an excess of serum in the pericardial sac, and is generally used to distinguish the passive secretion of dropsy from that of inflammatory effusions already described under Pericarditis. The pericardium naturally contains a very small quantity of serum, and after death from any cause it is common to find a few drachms of pale yellow fluid in it. When this reaches five or six ounces or more it constitutes dropsy of the pericardium, or hydropericardium. The causes of serous effusion, apart from inflammation, are those of general dropsy, such as Bright's disease, and such local interference with the venous circulation of the pericardium as valvular disease of the heart itself, chronic lung disease, and pressure of growths upon the veins which return blood from the pericardial surfaces. The liquid contained in the sac resembles that of dropsical effusion into the other serous cavities, being pale yellow, or more or less pink from exudation of blood-colouring matter, with a small quantity of fibrinogen, and from one to three per cent. of albumin.

The **Physical Signs** of hydropericardium are the same as those of effusion in pericarditis. As a rule, no special **Treatment** directed to the pericardium is required where the condition forms part of a general dropsy, or where it results from local interference with the circulation; the general dropsy or the valvular disease must be dealt with. In some rare cases the effusion may be so rapid or abundant as to require paracentesis of the pericardium.

PNEUMOPERICARDIUM AND PNEUMO-HYDROPERICARDIUM

These signify respectively the presence of gas, and the presence of gas and liquid together, in the pericardium. Gas in conjunction with liquid has been observed as a result (1) of decomposition of the liquid of pericarditis; and (2) of the communication of the pericardial sac with air-containing cavities. This communication may be traumatic, as in the case of a juggler, who, in attempting to swallow a blunt sword, perforated the pericardium from the œsophagus; as in the case recorded by Flint, where the pericardium was punctured by a stab through the pleura; and after the operation of paracentesis pericardii. Or the communication may be effected by disease; and cases are on record of cancer of the œsophagus ulcerating into it, of a phthisical cavity opening into it, and of a hepatic abscess communicating at the same time with the peri-

cardium and with the stomach. Gas can never be observed alone in the pericardium, as its entrance from without is almost immediately followed by pericarditis with liquid effusion.

The **Physical Signs** of pneumo-hydropericardium are resonance on percussion over the præcordial area, and splashing, churning, or gurgling sounds, synchronous with the movements of the heart. This last sign may be absent where the gas is largely in excess of the liquid; but in a case recorded by Walshe there was this peculiarity, that on placing the patient on one side the resonance was accompanied by an area of dulness due to liquid at the lowest part, and on turning him to the other side the resonance and dulness changed their relative positions. A few cases of recovery are recorded.

HÆMOPERICARDIUM

In slighter degrees, the effusion of blood into the pericardium occurs in so-called hæmorrhagic pericarditis, from the rupture of the new-formed vessels; but larger quantities, when not directly traumatic, result from rupture of the myocardium, of an aneurysmal sac, or of vessels in a cancerous growth. Scurvy and allied conditions may also give rise to pericardial hæmorrhage.

Symptoms.—When sudden effusion of blood into the pericardium takes place, the patient is seized with more or less oppression of the chest, pallor, syncope, unconsciousness and death in quick succession; or with the same pallor and with collapse, feeble pulse, dyspnœa, and orthopnœa he may remain for twenty-four or thirty-six hours before the fatal termination; or presumably, with a less degeee of hæmorrhage, death may be still further delayed and a pericarditis may develop, which contributes to the final result. Walshe refers to cases, probably of a scorbutic nature, or at any rate not dependent on rupture of aneurysms, or of the heart itself, in which recovery has taken place.

The **Physical Signs** are those of a large pericardial effusion; extensive præcordial dulness, and enfeeblement or absence of the heart-sounds. The **Diagnosis** would be assisted by a knowledge of the previous existence of aneurysm, or attacks of angina pectoris.

Treatment.—Absolute rest and judicious use of stimulants would give the only chance.

ANGINA PECTORIS

This name is given to an intense pain in the region of the heart, which comes on with great suddenness, and occasionally proves fatal.

Ætiology.—It scarcely occurs before middle age, and it is very much more frequent in men than in women, in the proportion of ten to one. As a large majority of the cases present some lesion of the heart or arteries, the conditions which lead up to these

changes may be regarded as predisposing causes of angina pectoris; and especially obesity, sedentary occupations, and the gouty habit. Heredity also seems to have an influence. The immediately exciting causes are mostly such as may be supposed to act prejudicially upon the functions of the heart, whether through its muscular or nervous apparatus. The most frequent are physical exercise, especially going uphill or against the wind, or moving about shortly after a meal; and emotional excitement, whether depressing or exhilarating. Much slighter exertion, or exposure to cold, is sometimes sufficient; and occasionally the attack begins during sleep.

Symptoms.—The patient is seized quite suddenly with acute pain in the front of the chest, over the third or fourth left costal cartilage, or over the manubrium sterni, or across the upper sternum from right to left, or over the heart. The pain radiates thence to the left side and back, or through to the scapula; up to the left shoulder, and down the left arm to the hand; or less frequently to the right shoulder, arm, and hand. Tingling or numbness may accompany the pain in the fingers, and with this there is a feeling of tightness of the chest, or suffocation, and even of impending death; but there is no dyspnœa of the usual type. The patient is obliged to stop if he is walking; he becomes collapsed, faint, and covered with clammy perspiration. The behaviour of the heart seems to be variable; at any rate accounts differ widely. The pulse is sometimes irregular; sometimes quickened, or quick and feeble; sometimes it stops altogether for a time. The tension may be increased at the time of the attack; but it is not so always. The attack is often accompanied by flatulence, and followed by the passage of abundant pale urine. After lasting a few seconds or minutes, the pain quickly passes off, but it may recur again frequently in the course of a few hours, or it may not be experienced again for several months or years. Angina may be fatal in the first and only attack. There is considerable variety in the onset of the pain in different cases: thus it may begin in the arm or arms and spread up to the chest; or it may begin in the upper abdomen. The attack may also be accompanied by acute respiratory disturbance, such as dyspnœa, cyanosis, Cheyne-Stokes breathing, or œdema of the lungs: and occasionally there are cerebral symptoms.

Slight attacks of cardiac pain occur in some patients, which have not the dramatic features of the case first described; they do not, however, differ materially from true angina. They depend on similar arterial or cardiac lesions, and are cured or relieved by the same means.

Pathology.—When death has taken place in an attack, the heart has generally been found relaxed, with its cavities full of blood. In the majority of cases, some disease of the heart or aorta has been found, and mostly of the following kinds: fatty degeneration of the myocardium; syphilitic aortitis, or atheroma, or aneurysmal dilatation of the aorta; atheroma or calcification or shrinkage of the sigmoid valves; and arterio-sclerosis or calcareous deposit

in the coronary arteries, or their obliteration from endarteritis or thrombus. Clinically, also, in a large proportion of cases, some auscultatory evidence of one of the above cardiac defects is forthcoming. Disease of the mitral orifice, on the other hand, is rarely, though sometimes, the sole cause of angina pectoris. And there are cases in which no lesion whatever of heart or vessels can be found.

The predominance of these lesions is one strong argument against the disease being purely neuralgic; but it is still an open question how the lesions produce the acute pain and the fatal result. The explanation commonly adopted is that a heart degenerated in consequence of the disease of the coronary arteries is put to a sudden strain, either by an increase in the peripheral resistance, or by the need for an additional effort; and that the strain or shock, or the over-distension, or spasmodic contraction of some of the ventricular fibres gives rise to the pain. Some writers see a resemblance between angina pectoris and intermittent claudication (*see* p. 661). Liégeois and others refer angina to an *ischæmia*, or anæmia of the heart's substance, consequent upon the actual constriction of the coronary arteries in some cases, and upon imperfect blood-supply through them in others —*e.g.*, free aortic regurgitation. Allbutt thinks that the pain is due to tension of the inflamed coats of the aorta (aortitis) above the sigmoid valves: and that death is caused by shock inhibiting the heart. A consideration of the tension of the pulse led Brunton to administer nitrite of amyl, with the object of diminishing it; and the angina was promptly cured. But the arterial tension is not always high, even in cases which are instantly relieved by vaso-dilators. Liégeois suggests that vaso-dilators do good by flushing the heart itself with blood.

Diagnosis.—The character of the pain, its occurrence as the result of exertion, and the evidence of a cardiac or arterial lesion (valvular disease or arterio-sclerosis), are generally conclusive. It has to be distinguished from neuralgic pains, especially in neurotic women, in whom actual lesions are not present, though the pain may be accompanied by signs of vasomotor constriction (coldness and numbness of the extremities), and from gastric disturbance which may occur at any adult age. In this case the pain often occurs during rest, lasts much longer than angina, and may be accompanied by tumultuous action of the heart, and palpitation.

Prognosis.—In its severer forms this must be grave, as there is always a fear of recurrence, which may be too quickly fatal for treatment to be of any avail. But milder cases are often amenable to treatment.

Treatment.—Undoubtedly the most efficacious remedy for an attack of angina is nitrite of amyl, which is conveniently carried in small glass capsules, each containing from three to five minims one of these is crushed between the folds of a handkerchief, and the vapour is inhaled freely. The effect is to dilate the peripheral arterioles; the face flushes, the cranial vessels throb, and the pain is often relieved at once. The dose may have to be repeated. A

similar effect may be obtained by the administration of nitro-glycerine internally, but its action is not so rapid. One may begin with $\frac{1}{100}$ minim, given in the form of tabella; or with a one per cent. solution in alcohol, of which 1 minim in a little water is the required dose. Much larger quantities may have to be given, equivalent to two, three, five, or ten-hundredths of a minim. The first administration of even small doses of nitro-glycerine is often followed by a throbbing headache, but after a time tolerance is established and the larger doses can be borne. Sodium nitrite ($2\frac{1}{2}$ gr. in tabella) and erythrol tetranitrate (1 gr. in 1 drachm absolute alcohol suitably diluted) are also good vaso-dilators. If these measures fail, the hypodermic injection of morphia, or the inhalation of chloroform, may be used; and much collapse will require brandy or ether. When angina has once declared itself in a patient, nitro-glycerine should be given for several weeks; the dose may be $\frac{1}{100}$ minim three or four times daily, gradually increased to $\frac{1}{20}$ or $\frac{1}{10}$. Iodide of potassium (5 to 30 grains) is also beneficial in some cases, especially where syphilitic aortitis may be suspected. Arsenic and iron are valuable as cardiac tonics. At all times undue exercise, sudden movements, excess in eating and drinking and smoking, and mental excitement should be avoided; and generally the treatment suitable to arterio-sclerosis should be pursued.

FUNCTIONAL DISORDERS OF THE HEART

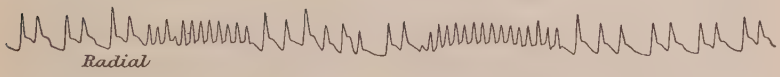
The functional disorders of the heart which we may here shortly consider, are frequent action or *tachycardia*, slow action or *brady-cardia*, intermission of the heart's beat, and palpitation. All these may be seen in various forms of cardiac disease, but are sometimes present temporarily, or in paroxysms, when no organic disease of the heart can be discovered.

FREQUENT ACTION, OR TACHYCARDIA

One may put aside as normal the high frequency of the pulse in some healthy persons, and the increased rapidity on exertion. Three important causes of increased frequency are: (1) organic disease of the heart, whether valvular or myocardial; (2) pyrexia probably acting through toxins; and (3) temporary nervous influences. It is always important to remember this, both when consulted for actual illness and when asked to examine a candidate for life insurance; the pulse in such persons may run up at once to 120 or 140, though they are at the same time in perfect health. Tachycardia results from paralysis of the vagus, and is thus seen in multiple neuritis; it is also a sequel of influenza, and is an important feature in Graves' disease. Apart from any of these causes, there occur from time to time cases of paroxysmal increase in the frequency of the heart's beat, which have many features in common.

Paroxysmal Tachycardia.—This condition occurs in both sexes and at all ages above early childhood. There is no constant antecedent. Some are in perfect health with no previous illnesses to record; some have already cardiac lesions; some have had syphilis or rheumatic fever. The first attack, or subsequent attacks, may

FIG. 65



An arterial tracing from a case of paroxysmal tachycardia. Two short paroxysms are shown, each lasting approximately five seconds. Between the paroxysms the pulse is irregular. (*After Lewis.*)

appear to be brought on by a strain, or injury, or emotion, or a fit of indigestion: often, however, the attack is spontaneous, and may even start during sleep. Often the change is quite sudden, and the heart's frequency runs up to 150, 200, or 250; figures which can sometimes be surely counted only by auscultation of the heart, or by graphic record, and not by feeling the pulse. The carotids pulsate, the heart's action may be a little more forcible than usual, but often the patient suffers very little discomfort. It may last a few minutes and then cease as suddenly as it began, and the pulse fall to 80 or 70 (*see* Fig. 65). On the other hand, it may last some hours, or a day or five or six days: and in these longer cases dyspnœa, dilatation of the heart, bronchitis, and œdema of the lung, enlargement of the liver and albuminuria may result, to disappear with the cessation of the tachycardia. With dilatation of the heart a temporary systolic murmur may be heard; on the other hand, in some cases of organic disease murmurs previously present may disappear in the attack. The arterial tension is usually lowered, and the urine is scanty. The attacks are little amenable to treatment, they may recur at longer or shorter intervals for years, and if frequently repeated in a severe form may be fatal.

By recent work upon these cases with the polygraph or string-galvanometer, two forms have been distinguished. In one, each auricular beat corresponds to and precedes a ventricular beat in the normal manner (*auricular form*). In the other, an auricular beat is not visible in the jugular tracing, whence it is inferred either that it takes place at the same time, or is preceded by the ventricular contraction; in other words, that the beat is initiated by the ventricle (*ventricular form*). The disturbance of function probably starts in the cardiac substance, perhaps in the bundle of His, or in the auriculo-ventricular node, and not in the nerve apparatus: the frequency is greater than any that could be due to a vagal lesion, and it is not affected by atropine or aconite.

Treatment.—Digitalis should be tried, though it has often failed. Compression of the thorax laterally is said to have stopped attacks: and pressure on the vagus may assist, even if this nerve is not primarily responsible. Electrical stimulation of this nerve in the neck, with the anode at the back of the neck and the kathode in front of the sterno-mastoid, has been also found useful. In some cases the tachycardia yields to morphine injections. One should try to prevent the return of the paroxysm by rest, by the avoidance of all excitement, by careful attention to diet, by abstinence from tea, coffee, and tobacco, and by a course of potassium or ammonium bromide in moderate doses.

INFREQUENT ACTION, OR BRADYCARDIA

This condition is also peculiar to some people throughout life. It accompanies sclerosis of the coronary arteries and some other lesions which have already been mentioned (p. 572), and occurs as a result of, or in the course of, numerous disorders, such as pain, collapse, exhaustion, convalescence from acute infectious disease, hysteria, mental disorders, cerebral tumours, jaundice, and many toxic states. It has been already shown that an infrequent radial pulse may be caused by alternate beats of the heart not being strong enough for the wave to reach the wrist, or by auricular contractions failing to reach the ventricle, the condition known as heart-block (*see* pp. 562, 624). A pulse constantly less than 40, in a person who has formerly had one of normal frequency is probably due to this. It is sometimes called *false bradycardia.*

The object of **treatment** should be the removal of the cause. For a direct influence upon the circulation, belladonna and massage have been recommended; but atropine appears to increase the frequency of the auricular contractions without affecting the ventricles.

INTERMISSION AND EXTRA-SYSTOLE

We have previously stated (*see* p. 573) that in the so-called intermittent pulse a premature beat or extra-systole occurs so close to its predecessor that it is not felt by the finger, and is usually followed by a long diastolic period, such that the interval between the two good beats is twice the usual interval. There is often at the same time a sudden and distressing sinking sensation in the cardiac region, or a feeling as if the heart fluttered or tumbled over. This may be a purely functional event, independent of any disease whatever of the heart. On the other hand, extra-systoles are of frequent occurrence in valvular and myocardial disease, when, being associated with other irregularities, they may fail to give rise to the sensation of a dropped beat or intermission.

Ætiology.—Intermission of this functional nature occurs at almost any adult age. In younger persons it is often traceable to gastric disturbance, or to the too free indulgence in tea, coffee, or tobacco.

Overloading the stomach is a common cause, and it frequently occurs in the early morning hours after a heavy supper or late dinner. In such a case it ceases as the stomach disposes of its contents. When the intermission is due to tea or tobacco, or repeated indigestion or flatulence, it is more persistent, and it may be present day after day until the cause has been discovered and removed. In persons past middle life, intermission is apt to be troublesome for long periods, or even to the end of life. In some cases the above-mentioned causes may be at work; in others there may be evidence of senile changes such as atheromatous arteries; in others, again, no appreciable alteration in the heart, arteries, or other organs can be detected, and the patient's capacity for exertion is perfectly normal.

The **Prognosis** is more doubtful in the case of older patients. Many people, it is true, live on without further indications of cardiac change; but in those with arterial degeneration the symptom must not be ignored.

Treatment.—In young people, tea, coffee, and tobacco should be forbidden if either of these can be shown to cause the trouble. All mental worry or physical overstrain should be avoided; the diet should be carefully attended to; the digestion should be assisted, and flatulence prevented, by bismuth, sodium bicarbonate, spiritus ammoniæ aromaticus, and calumba or gentian. Diffusible stimulants, such as ether, ammonia, and alcohol, are likely to diminish intermission for a time; but ammonia is the only one that can be safely used continuously.

PALPITATION

By this term is meant an unduly forcible action of the heart perceptible to the patient.

Ætiology.—As a functional disorder, it is especially common in nervous, hysterical females, but occurs in both sexes as the result of depressing influences upon the nervous system, such as mental worry and excitement, over-study, exhausting illnesses, anæmia, and excessive smoking or tea-drinking. It is paroxysmal in its occurrence, and the attacks may be induced by excitement, physical exercise, overloading of the stomach, or indigestion.

Symptoms.—During a severe attack there is much distress from the violent beating of the heart against the chest-wall, the carotids throb, there is a sense of faintness, or a fear of impending death. The heart's impulse is in the natural position, but the beat is widely diffused; the præcordial dulness is not increased, and the pulse is not necessarily quicker than normal. The cardiac sounds are loud, clear, and ringing, and there is no murmur except in cases of anæmia. The attack may last from some minutes to a few hours, and is occasionally followed by the passage of a quantity of pale urine.

Treatment.—This must be in great part preventive. The general health should be as far as possible improved, the patient should avoid all physical and mental excitement, and should abstain from tea, coffee, alcohol, and tobacco. Any digestive disturbance

should be corrected. During the attacks digitalis should be given in doses of 5 or 10 minims of the tincture every two or three hours, combined with ammonium or potassium bromide; or with valerian, asafœtida, or spirits of nitrous ether in the usual doses.

DISEASES OF THE BLOOD-VESSELS

The diseases of arteries are mainly comprised in inflammation and degeneration, and the mechanical results of those lesions. Inflammation of the veins, or phlebitis, and thrombosis and embolism will also be considered in this section.

ARTERITIS

Ætiology.—The following are recognised causes of arterial inflammation: (1) Infections acting directly, as contact of the artery with inflammatory, septic, or suppurative foci; impaction in the artery of septic emboli. In a tubercular lung, the arteries are directly invaded from without by tubercle; and in tubercular meningitis tubercles form on the minute vessels of the meninges, the infection being probably lodged in the walls from the circulating blood. (2) Toxins and poisons circulating in the blood, such as those of syphilis, lead, alcohol, and gout; (3) overstrain, which acts especially as a cause of inflammation of the aorta and large vessels.

Anatomical Changes.—Inflammation of the arteries may be acute or chronic, local or general. *Acute local arteritis* is seen in the above-mentioned instances of direct infection, when a vessel is in contact with a wound or abscess, or is the subject of embolism; the outer or inner coat in this case is infected with organisms, inflammatory changes take place which result in softening, yielding (aneurysm), or perforation (with hæmorrhage) of the arterial wall. In malignant endocarditis, the inner coat of the aorta is often infected from the valves, and undergoes the same pathological processes. A *general acute aortitis* also occurs, in which all the coats of the vessel become thickened with cellular infiltrations, especially about the vasa vasorum, as well as on the surface, and in the meshes of the inner coat. The aorta or any other artery affected in this way becomes weaker in its resisting power, and is liable to dilatation from the pressure of the blood within.

In *chronic arteritis*, which may begin as an acute disease, the inner surface presents broad grayish-white slightly raised patches, which may be soft, mucoid, and gelatinous, or more or less sclerosed from the presence of fibrous tissue. In the later stages there may be a semi-cartilaginous thickening, affecting the inner coat mainly. But the same changes involve also the middle coat, destroying the muscular and elastic elements; and the outer coat, causing fibrous

thickening and condensation of the tissue. In the small and middle-sized arteries the thickening of the intima causes considerable diminution of the lumen of the artery (*endarteritis obliterans*). It begins with cell-proliferation, which is succeeded by fibrous transformation; the adventitia is also affected, but the media much less so. This condition not infrequently results from syphilis, and may lead to thrombosis at the narrowed part, as is often observed in the cerebral arteries.

Symptoms.—Acute aortitis is often unaccompanied by symptoms, but it may cause pain behind the sternum, radiating to the arms, or definite anginal attacks. In some cases of acute arteritis, affecting the vessels of the extremities, there have been pain and tenderness, limited to the course of the vessels, gradually spreading down the limbs to the extremities; and in others, pain, tenderness, loss of pulse in the affected vessels, gangrene of portions of skin in the area corresponding to their distribution, or shedding of the nails. General arteritis has also been seen in a chronic form, leading again to pains in the limbs along the course of the arteries, and followed by obliteration or such narrowing of the channel as to abolish the pulse. But as a general rule the effects of arteritis are only manifest in the degenerative changes which may be felt in accessible arteries, or they reveal themselves in the symptoms of aneurysm, thrombosis, or renal disease.

ARTERIAL DEGENERATIONS

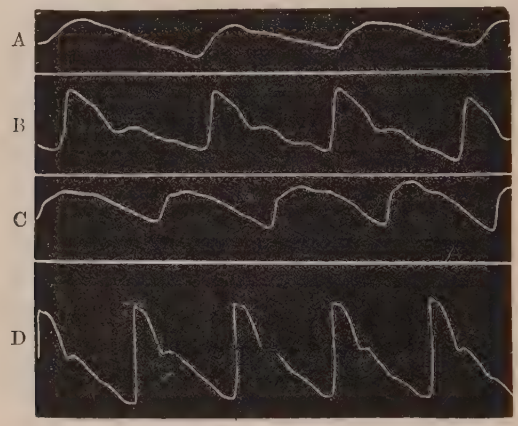
Arterial degeneration occurs in three forms, *atheroma*, *arteriosclerosis*, and the *amyloid* or *lardaceous* change. The first two terms are given to arterial changes, which are closely allied and frequently occur in the same subjects, but which, presenting somewhat different histological features, may probably with justice be kept apart. They are both common in middle age and advanced life; and they arise from various chronic intoxications, such as those which form part of gout and chronic Bright's disease (whether these be of intestinal or other origin), from syphilis, alcohol, and probably sometimes malaria, acute infections, diabetes, and the excessive use of tobacco. The circulation of poisons in the blood is here the efficient cause of the changes in the vessel walls. Excessive strain of manual or other labour, as well as prolonged mental and moral strain, according to some, acting through the vasomotor system, can also probably induce them.

Syphilis is an important factor in atheroma: while the amyloid change is chiefly due to the toxins of syphilitic and of pyogenic organisms. This last form of degeneration will be described under Lardaceous Disease of the Liver.

Atheroma as a senile change is probably of purely degenerative nature, causing a fatty change in the cells of the innermost layers of the intima; but it often follows upon definite inflammatory

changes such as those which have just been described. Thus the gray semi-cartilaginous patches become mixed with others of a yellow colour, which arise from a fatty change taking place in the inflamed tissue. A pasty mass results, which contains fat granules and cholesterin crystals. It affects chiefly the deeper layers of the

FIG. 66



A. Right Radial Pulse in a Case of Aortic Aneurysm compressing the Right Innominate Artery.
B. Left Radial Pulse in the same Case.
C. Radial Pulse in Compression of the Subclavian Artery.
D. Atheromatous Artery. Pressure, Six Ounces.

inner coat, but may approach to the surface, so that the innermost layers finally give way, and an *atheromatous ulcer* results. In some cases calcareous granules are deposited and form plates or spicules, upon which fibrin may be deposited from the circulating blood. The combination of patches of early inflammation, atheroma and calcareous deposits, with the irregular dilatation of the vessel which occurs as the various weakened spots yield to the blood-pressure, constitutes *endarteritis deformans*. It affects, first of all and more constantly, the aorta and the larger arteries: in the senile forms the arteries are dilated, tortuous, and rigid. There is extreme degeneration of the media, and calcareous plates are deposited on the intima.

While atheroma occurs in patches, with a very irregular localisation in different parts of the body, *arterio-sclerosis* is much more uniformly distributed, and may affect all the coats in varying degrees. Sclerosis of the intima, a uniform fibrous thickening of the inner coat, is found in middle-sized and smaller arteries. Both this and sclerosis or fibrous thickening of the adventitia are sometimes secondary to sclerosis of the media or muscular coat. This last may, however, exist by itself, and constitutes the main feature of arterio-sclerosis at an advanced age. It is a hypertrophy of the muscular elements, to which may be added in later stages granular

degeneration or necrosis, and less commonly calcification. It is probably often the result of increased arterial tension, and in many cases is accompanied by granular disease of the kidney. But it is still an open question whether the granular kidney is the cause or the result; or a common result with arterio-sclerosis of one of the chronic intoxications above mentioned.

Symptoms.—*Atheroma.*—When at all extensive, atheromatous degeneration can be recognised by its effects upon those arteries which are accessible to examination during life. First, the artery-wall becomes rigid; secondly, yielding to the pressure of the blood-current, the walls become stretched, and make the vessel both larger in its transverse diameter, and at the same time longer; thirdly, the increased length is accommodated by the vessel becoming tortuous instead of nearly straight. At each beat of the heart the artery is not only expanded, but bent still more out of a straight course. To the finger the artery feels hard, rigid, and rough, whilst the pulse consists of a sudden expansion, and a slow, almost unbroken collapse. Fig. 66, D, shows the characters of a sphygmographic tracing; the pressure is not necessarily high.

It can hardly be said that any general condition of illness necessarily accompanies atheromatous arteries. Many persons in advanced life with this form of degeneration are in the enjoyment of perfect health. But such diseased vessels frequently co-exist with chronic Bright's disease; by their want of elastic tissue they tend to hypertrophy and dilatation of the heart; vertigo, headache, and shortness of breath often occur in connection with them; and they lead to more serious results in several ways, by rupture (cerebral hæmorrhage), by thrombosis and obstruction (hemiplegia, gangrene of the limbs, fatty degeneration of the heart, angina pectoris, and Adams-Stokes disease), or by local dilatation (aneurysm).

Arterio-sclerosis.—The sclerotic artery is more uniformly firm to the touch than the atheromatous artery, less likely to be dilated, and more generally accompanied by high arterial tension: which may give a characteristic sphygmographic tracing with a pressure of from five to twelve ounces, and a reading on the sphygmomanometer of from 150 to 260 mm. Hg. The aortic sound is accentuated, and the first cardiac sound is prolonged or doubled. There are, however, cases of arterio-sclerosis with diminished arterial tension, and this is sometimes due to increased tension in the portal system (Huchard).

The subject of the symptoms of arterio-sclerosis presents the difficulty that, since the condition is almost certainly brought about in most instances by the action of toxins or poisons circulating in the blood, which produce excessive tension in the vessels, the toxæmia and the increased tension are as likely to be responsible for the symptoms as the structural change in the arterial walls. Since, however, the latter, or sclerosis, probably helps to perpetuate the increased tension, it may be allowed a share in such results as are not due to the toxæmia.

The general symptoms occurring in gouty persons, and the early symptoms of chronic Bright's disease are largely toxic in origin, such as headache, insomnia, and dyspnœa: but in both of these conditions high tension is present.

Eventually the arterial lesions, whether sclerosis or high tension, have their effect upon the heart, which, struggling to overcome the arterial resistance, becomes at first hypertrophied, then dilated, then irregular in its action with or without the murmur of regurgitation through the valves: and the patient suffers from all the results of dilatation and incapacity of this organ (asystole)—dropsy, dyspnœa, albuminuria, &c. Arterio-sclerosis in particular organs may be accompanied by local symptoms, due probably to the limitation of the circulation—in the heart itself angina, in the brain symptoms of softening or hæmorrhage, such as forgetfulness, confusion of thought, or paralysis. Sclerosis of the renal arteries is a constant associate of chronic interstitial nephritis.

Treatment.—Little can be done for the treatment of arterio-sclerosis itself. Attempts may be made to influence the source of intoxication which is causing it: especially by the avoidance of butcher's meat and highly nitrogenised foods or those containing purin-bodies, abstention from alcohol, tea, and tobacco, and from all excessive mental or physical strains. If arterial tension is high a strict milk diet, or one from which chlorides are excluded, may be desirable: and diuretics such as the salines, and theobromine (1 to 5 grains) with thyminic acid (2 or 3 grains). The effects of vaso-dilators like nitro-glycerine, or sodium nitrite, may also be tried; and massage, muscular exercises, high-frequency currents, and hydrotherapy will sometimes be beneficial. Digitalis is not suited for early stages, but may be helpful when the heart has got to an advanced stage of dilatation. With dilatation of the right ventricle and pulmonary œdema venesection may be employed.

ANEURYSM

This name (*ἀνευρύνω*, to widen) is applied to dilatation of an artery for a more or less limited extent of its course. Aneurysms are divided, according to their shape, into *fusiform* and *sacculated;* the fusiform being a more or less uniform dilatation of the whole circumference of the vessel; the sacculated forming a globular projection from one side of the vessel, and connected with it in advanced cases by a constriction or neck. They have been divided also into *true* and *false,* according to the number of the arterial coats still present in the sac; but it appears that the inner coat never persists in aneurysms above a certain size, and hence this distinction has no apparent value. Sometimes, especially in the limbs or the abdomen, a sacculated aneurysm ruptures at a prominent point, blood oozes slowly out into the tissue around and forms a coagulum, bounded by a kind of cyst of inflammatory tissue. This

has been called a *diffused* aneurysm. Lastly, a *dissecting* aneurysm is formed when at a part of the artery affected with atheroma the blood penetrates the inner and middle coats, and forces its way between them and the outer coat.

Ætiology.—Aneurysms arise from any cause that weakens the vessel at one point. The most common cause is atheroma, especially in the large vessels, in which the inner and middle coats are weakened, and the whole wall yields to the pressure of the blood at that point. In smaller vessels, such as those of the brain and lungs, the vessel may be weakened by the local causes of arteritis already mentioned—viz., embolism, or the invasion of tubercle. Surgical injuries of the outer coat also lead to aneurysm. Of the more general causes disposing to aneurysm, syphilis holds an important place; and probably also excessive muscular strain acting through the circulation.

Results.—These are mostly seen in the sacculated forms. One result is the *coagulation* of blood in the sac itself. As this is out of the direct current it moves more slowly, and its coagulation is favoured by the roughness of the aneurysmal sac. The sac thus becomes lined, or nearly filled with successive layers of pale buff, fibrinous deposits; and it is by the complete filling of the sac with these fibrinous layers that aneurysms may be obliterated and cured. The greater the freedom of communication with the main vessel, the less the liability to the formation of fibrin; and in a fusiform aneurysm no deposits take place.

Another result of aneurysm is its *pressure* upon the parts around it. The sac may attain an enormous size; an aneurysm, of which there is a model in the Museum of Guy's Hospital, springing from the aortic arch, measured eight inches in diameter. As it enlarges, the growth presses with irresistible force upon adjacent parts, displacing the various organs, compressing and obstructing the blood-vessels, the trachea, the bronchi, or the œsophagus, flattening and stretching nerves, and causing thereby pain, numbness, or paralysis, according to the nerve involved, and the degree of its compression. When it comes in contact with unyielding bone, an aneurysm causes absorption of the osseous tissue, and excavates or actually perforates it. The vertebræ are frequently eroded in this way, and it is remarkable that the intervertebral cartilages are more resistant than the bone, so that when the aneurysm is large enough to cover more than one vertebra, the cartilage projects between the two cavities which have been made in the adjacent bones. The ribs and sternum, at first slightly raised by the advance of an aneurysm, are subsequently perforated and allow the pulsation of the tumour directly under the skin. Analogous results occur in other parts of the body, but it is chiefly in the thorax that the pressure-effects of aneurysm are manifested, because the bony walls allow no room for their important contents to escape.

A third effect of aneurysm is *hæmorrhage*, which is the cause of death in a large percentage of cases. The great distension of the

coats, and the degeneration which precedes this, sufficiently explain why hæmorrhage occurs. Even the deposit of layers of fibrin within the sac will not always prevent it; the clot, if at all abundant, does not organise, and the blood may force its way into fissures and meshes of the coagulum, and so finally reach the surface, and slowly ooze out. The rapidity and extent of the hæmorrhage further depend upon the support the aneurysm has from without. Ruptures into hollow viscera and serous cavities are often rapidly fatal; though I have known a man live ten days after a rupture with hæmorrhage into the pleura. Ruptures into connective tissue or inter-muscular spaces are often much slower in their effects, and in the limbs may allow time for successful treatment.

Symptoms.—They may be divided into those common to all aneurysms, and those determined by the locality; of the latter only the special symptoms of thoracic aneurysm will be now considered, those characteristic of abdominal aneurysm being discussed elsewhere (*see* Abdominal Tumours).

The symptoms common to aneurysm in any part of the body are: (1) Tumour; (2) Pulsation; (3) Murmur; (4) Pain; and (5) Other effects of pressure.

Tumour, or some kind of swelling, is a necessary part of an aneurysm, but it may, of course, be entirely unrecognisable during life in such parts as the cranium, the thorax, the deeper parts of the abdomen, or the gluteal region.

Pulsation is the characteristic symptom, showing the connection of the tumour with an artery. It is nearly synchronous with the cardiac systole and the radial pulse, mostly rather slow and heaving, expansile—that is, enlarging the tumour in all directions at once, and not merely in a direction perpendicular to the course of the artery apparently affected, which would be the kind of pulsation communicated by an artery to an independent tumour over it. Pulsation is affected by the amount of coagulum lining the sac, and is in some cases absent.

Murmur.—On listening with the stethoscope over an aneurysm in the abdomen or limbs a murmur is mostly audible, synchronous with the beat of the pulse, varying in quality, soft, harsh, or loud. It is due to the passage of the blood from the normal artery into the wide cavity of the sac. In some cases a diastolic murmur is present; and with a feeble current of blood, or with much coagulum in the sac, and perhaps under other circumstances, the murmur may be absent.

Pain is frequently present, and results from the stretching of, or pressure on, parts in the neighbourhood. It occurs at the seat of the aneurysm, and often radiates in different directions from it—to the shoulder and down the arm, for instance, in thoracic aneurysm, and along the course of the intercostal nerves when the spine is eroded.

The other effects of pressure will vary with the seat of the aneurysm.

ANEURYSM OF THE THORACIC AORTA

Aneurysm may occur at any part of the thoracic aorta, from the sigmoid valves to the diaphragm; but the first part is by far the most frequently involved, and here especially all varieties occur, from irregular dilatations of the whole calibre to true sacculated aneurysms.

The results of the former, *dilated aorta*, are not seen so much in the pressure effects. If the part adjacent to the valves is affected, the orifice may be dilated, the valves are thus rendered incompetent to close it, and regurgitation will take place, followed by hypertrophy and dilatation of the left ventricle. The symptoms are chiefly those of aortic regurgitation. In other cases of dilated aorta there is a systolic murmur, and the patient suffers from attacks of severe cardiac pain, with many of the characteristics of *angina pectoris*, on anything beyond the most moderate exertion. Such patients sometimes die suddenly.

A *sacculated aneurysm* causes symptoms which depend on its position and the direction of its growth. In the first part of the arch it may grow forwards and present itself as a pulsating tumour in the second or third right intercostal space, less commonly in the second or third left space. The tumour is slightly tender, often the seat of pain, which is aggravated by exertion; and commonly a soft systolic murmur is heard over it. Growing towards the right, a tumour in this region presses upon the superior vena cava, causing œdema of the arms, or it grows into the upper part of the right chest, compressing the upper lobe of the right lung, or the bronchus leading to it, and producing deficient breath-sound and at a later stage dulness over the corresponding area. Towards the left an aneurysm may press upon the pulmonary artery, cause dilatation of the right heart, and ultimately open into the pulmonary artery. Rupture into the pericardium is a not infrequent termination of aneurysms in this situation.

In the second part of the arch, an aneurysm especially affects the convex border at the site of the origin of the great vessels, and frequently grows upward to the base of the neck, where it forms a pulsating tumour, and is with difficulty distinguished from aneurysm of the carotid or innominate artery. The pressure effects, if present, are mainly directed towards the trachea, producing stridulous breathing and dyspnœa; while the tumour itself is indicated by dulness over the upper end of the sternum, and by murmur. A large aneurysm in this position may drag the larynx downwards and to the left by pressure on the trachea. Aneurysms arising from the concave part of the arch come into relation with the left bronchus, which winds round it. Pressure on the bronchus causes in the corresponding lung diminution of the movement of the tidal air, in some cases early distension of the lung sufficient to displace the heart and depress the diaphragm; in all cases ultimately collapse

of the lung with retention of bronchial secretions and bronchiectasis. The physical signs are absence of vesicular murmur, with exaggerated breathing on the opposite side, hyper-resonance on percussion, later dulness with loss of voice sounds and of tactile vibration, later again localised bronchial or cavernous breathing, and moist râles (*see* pp. 493, 498). Compression of the lung itself may occur, with localised dulness and loss of respiratory murmur; and in some cases gangrene. Another result of pressure on the bronchus is that when, with the patient in the upright position, his mouth closed, and his chin raised to its fullest extent, the cricoid cartilage is grasped by the finger and thumb and gently lifted, the pulsation of the aneurysm is conveyed to the fingers holding the cricoid (W. S. Oliver). This sign, called *tracheal tugging*, has been said positively to indicate an aneurysm of the transverse arch; but an aneurysm adherent to the trachea may cause it as well as one pressing on the bronchus. Slight tugging can be felt in some healthy persons, but pronounced movement is a valuable sign of aneurysm. Pressure on the recurrent laryngeal nerve causes abductor paralysis of the left vocal cord, with subsequent "paralytic contracture" of the adductor, so that the cord occupies the middle of the glottis, and a certain amount of stridor and clanging cough may be the result.

Aneurysms of the aorta below the arch may press upon the œsophagus and cause dysphagia, or regurgitation of food; salivation after food has been noticed in some cases. Directed backwards, an aneurysm erodes the spine, presses upon intercostal nerves so as to cause intense pain, and later causes paraplegia by implicating the spinal cord. A murmur is often heard over the spine behind under these circumstances. Growing laterally the aneurysm may compress a large bronchus, or the lung.

Some other symptoms may be mentioned which are common to two or three of the situations discussed.

Inequality of the radial pulses.—The radial pulses may be unequal in volume, in sharpness of rise, and in blood-pressure. If the aneurysm compresses the innominate or the left subclavian artery, or obstructs either of these vessels at its origin by the formation of coagulum, the corresponding pulse is smaller, and less sharp in its rise, and shows in the sphygmographic tracing a sloping upstroke, or even complete absence of the percussion wave (Fig. 66, A, B, C). The blood-pressure as measured by the hæmomanometer is usually slightly above the normal in aneurysms of the aortic arch or innominate artery; but as a rule higher in cases of dilated aorta than in saccular aneurysm. A difference between the pressures in the two radial pulses of 5 or 10 mm. Hg. is common in these conditions, but also not infrequent in arterio-sclerosis. A constant difference of more than 20 mm. is in favour of aneurysm; but a difference of this degree was found only in 10 per cent. of cases (two out of twenty) of such aneurysms (O. K. Williamson).

Inequality of the pupils (*anisocoria*) has usually been attributed to

Skiagram of an aneurysm of the descending part of the arch of the aorta, showing the prominence to the left of the middle line and the oblique position of the heart in consequence of the pressure of the aneurysm. (Taken by Dr. A. C. Jordan.)

[*To face p.* 653.

interference with the sympathetic nerve fibres. Drs. Wall and Walker show reason for doubting if this explanation will meet every case; and they attribute the inequality to local pressure on the vessels, pointing out that normally pressure on the carotid will cause dilatation of the pupil of the same side.

Loss of flesh, cough, and dyspnœa on exertion or in paroxysms, are the common accompaniments of thoracic aneurysm of any size. Death takes place by exhaustion from pain and malnutrition; by interference with necessary functions from pressure upon such parts as the œsophagus, trachea, or bronchus; by inflammatory and septic processes from pressure on the lung; and, lastly, by rupture of the sac and hæmorrhage, either externally through the skin, or into the œsophagus or pericardium, or pleural sac.

Diagnosis.—The diagnosis must be made from a careful consideration of the above facts, remembering that the symptoms vary with the seat of aneurysm. Some of the combinations which may give rise to suspicion of aneurysm may be enumerated: (*a*) Pain like that of angina, with systolic murmur over the base of the aorta; (*b*) systolic murmur occurring at a part of the chest not corresponding to the seat of any valvular orifice; (*c*) pulsating tumour in or near the præcordial region; the points at which a normal or enlarged heart may beat must be remembered, as well as the fact that pulsation of the top of the right ventricle in the second left interspace is quite common; (*d*) obstruction of one or other radial pulse; but, of course, one or other subclavian artery may be obstructed by other means, so as to produce a local murmur and a delayed pulse; (*e*) obstruction of veins; (*f*) obstruction of trachea, bronchus, or œsophagus; (*g*) paralysis of one vocal cord, especially the left. The diagnosis may now often be confirmed by the use of the Röntgen rays (*see* Plates VII. and VIII.); and if dysphagia is the only symptom the question of aneurysm should be determined by X-rays, if possible, before the passage of a bougie, which might produce a fatal rupture.

Aneurysms near the base of the aorta are more likely to be confounded with valvular disease; those in the deeper parts of the chest with new growth (*see* Mediastinal New Growths). The difference has been stated in another way—namely, that aneurysms of the first part of the arch cause more physical signs, those of the transverse arch, more symptoms. The extremely rare pulsating pleurisy or empyema has been referred to (*see* p. 351).

Prognosis.—This is very unfavourable in the case of thoracic aneurysms. Complete consolidation with cure is rare; relief of symptoms and postponement of the fatal result for some years may be often effected.

Treatment.—The objects of treatment are to lessen enlargement, to prevent rupture, and to favour coagulation of blood in the sac. Everything which will quiet the circulation and diminish the force of the heart's contraction will act favourably in these directions. The surgical methods employed in aneurysms of the limbs are rarely

applicable in the case of thoracic aneurysms. Proximal ligature of the vessel is out of the question, and distal ligature or compression can only be applied to branches such as the carotid and subclavian. Insertion of horsehair, wire, or needles, and the injection of astringent fluids into the sac have had too little success in aneurysms of the limbs, where the operation is more under control, to be recommended for aortic aneurysms. Galvano-puncture has, perhaps, been more successful. Two needles, connected with the poles of a battery, are introduced into the sac of the aneurysm; on the passage of the current fibrin is deposited on the poles, but the danger of portions being detached, and causing embolism in remote parts, is present here, as it is in the methods just mentioned. The treatment of aneurysm in the chest generally resolves itself into: (1) rest; (2) restricted diet; (3) the use of anodynes and sedatives; (4) iodide of potassium. Rest must be absolute, the patient being in the recumbent position, and not allowed to sit or stand up for any reason whatever. The diet which was recommended by Tufnell, who strongly advocated this plan of treatment, consisted per diem of ten ounces of solid, including three of meat, and eight ounces of fluid, divided into three meals; but it is extremely trying, and few patients will submit to it. Opium or morphia is generally given to ease pain, to produce sleep, or calm restlessness, but other sedatives may also be of use, such as bromide of potassium, chloral, paraldehyde, or sulphonal. Pain may also be relieved by belladonna applications, or by cold; or by venesection to a small amount. Iodide of potassium appears to have a special influence upon the coagulation of blood in aneurysms, as great improvement in diminution of pulsation and of pain has followed its use, even though unassisted by restriction of diet. It should be given in increasing doses, up to 60, 90, or 100 grains daily. Gelatin in solution has been injected subcutaneously with some success, but also with so many accidents (*e.g.*, tetanus) as to discredit it seriously. From two to six ounces of a two-per-cent. solution in normal saline, carefully sterilised, are injected into the gluteal region every five or six days up to thirty or forty injections: the patient must be kept strictly at rest. Where the vocal cords are implicated by pressure on the recurrent laryngeal nerve, tracheotomy may be required to prevent fatal asphyxia. But aneurysms also cause dyspnœa by tracheal compression, for which tracheotomy would afford no relief.

PHLEBITIS

Inflammation of the veins, or phlebitis, results in thickening and infiltration of the walls with leucocytes, which may be in such numbers as to constitute a real suppuration of the coats. The terms *endophlebitis* and *periphlebitis* have been used to indicate inflammation of the intima and the adventitia respectively. Periphlebitis arises from contact with inflammatory foci outside the vein, or from

Skiagram of a large aneurysm of the descending part of the arch of the aorta, taken from the back. (Taken by Dr. A. C. Jordan.)

[*To face p.* 654.

injury. Endophlebitis is most often set up as a result of thrombosis, or coagulation of blood in the vein itself. This occurs from a variety of causes (*see* Thrombosis). The clot may then adhere to the vein-wall, becoming at the same time organised, and the vein may be completely obliterated. On the other hand, the clot may become channelled, and allow the continuation of the circulation; or in other cases it softens down into a puriform fluid. Periphlebitis extending inwards itself leads to thrombosis; on the other hand, abscesses may form in the tissue around the vein.

Symptoms.—Phlebitis is accompanied by pain and tenderness in the course of the affected vessel, with some reddening of the surface in the case of superficial veins. The vein can be felt as a prominent hard cord, and a varying amount of febrile reaction accompanies the local disease. The formation of abscesses will be indicated by hardening of the surrounding tissue, redness and œdema of the skin, and subsequently fluctuation. The secondary effects which result from breaking down, and transportation of the particles of thrombus, are described below.

The **Treatment** of phlebitis consists in complete rest of the part affected, the application of warm fomentations or of glycerine and belladonna to ease pain, and the administration of opiates, if necessary, for the same purpose. The risk of detachment of a thrombus must always be borne in mind (*see* Thrombosis and Embolism). If abscesses form the pus will have to be evacuated by incision.

THROMBOSIS AND EMBOLISM

Thrombosis is the name applied to the coagulation of blood within living vessels, whether arteries or veins, or in the cavities of the heart; and the clot itself is called a *thrombus*.

Embolism means the transference of a portion of clot or other substance (particles of tumour, parasites, fat-globules) from one part of the circulation to another, and its impaction when it arrives at a vessel too narrow for its further progress. This can only take place in the arteries (and portal veins), since the flow of blood in the veins generally is from those of a smaller to those of a larger calibre. The transferred particle is called an *embolus*.

Besides the conditions of fibrin-formation which usually determine coagulation, two important factors in *thrombosis* are undue slowness of the current of the blood, and some irregularity on the lining membrane of the vessel or cavity concerned; but it must be allowed that there is often an intimate relation with infective disorders, and it has still to be made clear what share either micro-organisms or toxins may have in the origination of the process.

Thus we see that blood coagulates in the heart upon its inflamed valves, or in its cavities when dilated or contracting with extreme feebleness. It coagulates in the vessels if their walls are injured, or are in connection with septic or gangrenous processes; in the

arteries especially when their walls are the subject of syphilitic or atheromatous lesions, or of aneurysmal dilatations; in the veins when the current of their blood is slowed by pressure, and with the slightest local disturbance in the subjects of various infective, cachectic, and anæmic disorders. The first step in the process seems often to be the accumulation of blood platelets at the determining spot, and later an aggregation of leucocytes, or the formation of fibrin. The effect of the coagulation of blood in a vessel is naturally to cause an obstruction, which will have different effects according as it is in an artery or in a vein. The coagulum once formed receives further deposits of fibrin from the blood circulating above and below, and so the thrombus may extend into larger and larger vessels. When first formed it is soft and fills the veins; but after a time it may shrink, and thus allow the re-establishment of the circulation. But the termination is not always so favourable. The thrombus commonly sets up some endarteritis or endophlebitis, adhesion to the wall of the vessel takes place, and ultimately the clot becomes organised, with permanent obliteration of the channel. In other cases septic micro-organisms may cause the coagulum to break down into a puriform fluid, which consists of pus-corpuscles, micrococci, and fine granular particles. An important result of thrombosis in the heart and in the veins is the detachment of fragments from the coagulum, and their transference to other parts of the circulation. They then become *emboli*, as above stated. The results differ according to the position and character of the original thrombus. Portions detached from venous thrombi are carried by the current of blood into the right auricle, thence into the right ventricle, and into the pulmonary artery, which they may block according to their size, either quite at its commencement, or in the substance of the lung. Thrombi in the right side of the heart will similarly cause embolism of the pulmonary artery; but thrombi on the aortic or mitral valves will cause embolism of the systemic arteries in the brain, spleen, kidneys, limbs, or elsewhere.

The result of embolism, unless a collateral circulation be promptly established, is the death of the tissue within the area of the vessel obstructed. The portion of tissue so affected is called an *infarct*. In the solid organs it usually has a conical shape and is therefore triangular or (so-called) wedge-shaped on section; and as seen *post-mortem* it is either hard, of white or yellowish-white colour (*white infarct*), or softer and blood-red in colour (*hæmorrhagic infarct*). In the former case the change is chiefly one of *coagulative necrosis;* the tissue deprived of its blood-supply is permeated with lymph from the surrounding living tissue, and coagulative changes take place in this. If the coagulable material is sufficient the infarct is hard, as seen in the kidney and spleen; if it is less abundant the infarct is softer, as in the brain. The white infarct is sometimes surrounded by a narrow margin of hæmorrhage. In the hæmorrhagic infarct the first process is also one of coagulative necrosis;

but to this is added more or less complete hæmorrhage by diapedesis of red corpuscles. Infarcts of the kidney and retina are commonly of the white variety; those of the lung and intestine are constantly hæmorrhagic; those of the spleen and heart may be of either kind. While infarcts in early stages are often somewhat swollen and project on the surface of the tissues, they subsequently, if not septic, become shrunken and contracted, as seen especially in the kidneys; the elements undergo fatty degeneration and are replaced by connective tissue. If the embolus comes from a suppurating thrombus, or is the product of malignant endocarditis, then the contained organisms may set up septic processes in the infarcts. These become purulent in the centre, forming abscesses, such as occur in the lungs in pyæmia; or in the brain and kidney occasionally in malignant endocarditis. Septic emboli, by infecting the arterial wall, sometimes determine the weakening and dilatation of the artery at the seat of impaction, and so the formation of an *embolic aneurysm*. If the main vessel of a peripheral part (foot, leg, or hand) is obstructed, to which no surrounding living tissue can supply coagulable material, the result is not coagulative necrosis, but gangrene.

The following are the more usually recognised forms of thrombosis and embolism:

Femoral Thrombosis.—This arises in the last stage of phthisis, cancer, and other exhausting diseases, in convalescence from typhoid fever and influenza, and after confinement (*phlegmasia alba dolens*). The leg becomes swollen, and the vein can be felt to be obstructed; there is generally also some tenderness from co-existing phlebitis. The detachment of a portion of clot followed by its impaction in a large branch of the pulmonary artery with sudden death, is an occasional accident.

Jugular thrombosis, and thrombosis of the *lateral sinus*, result from disease of the internal ear, or mastoid cells. From contact with the external ear, septic organisms are frequently present, severe phlebitis is set up, and the clot becomes septic. Particles are then conveyed through the right side of the heart to the lungs, in which pyæmic abscesses are formed (*see* p. 162). Other cerebral sinuses (longitudinal and cavernous) are sometimes thrombosed as a result of more general conditions, such as marasmus in infants, and chlorosis and anæmia in adults (*see* p. 363).

Thrombosis of the *pelvic veins* arises from disease of the pelvic viscera in women, or from gonorrhœa in both sexes.

Large clots sometimes form in the *heart*, just previous to death, when the circulation is failing, and in recesses of the walls in cases of dilatation. They may hasten death by hampering the action of the organ, or they may supply emboli to the pulmonary or systemic circulation.

Embolism and thrombosis of the *cerebral arteries* are described under Diseases of the Brain (p. 335).

Embolism of a large artery in a *limb* is not a very common event.

It causes sudden acute pain, followed at once by numbness, coldness, and loss of power in the limb: the pulse is imperceptible below the seat of embolism, and, as already stated, gangrene may result. In the *spleen* and *kidneys* the occurrence of embolism is not so commonly recognised. Sometimes there is sharp pain in the left side from embolism of the spleen. Embolism of the kidney causes frequently albuminuria, with perhaps blood in the urine; and in malignant endocarditis there is often double nephritis, probably from minute emboli (*see* p. 583). Cases of embolism of the *mesenteric artery* have occurred in which the patient has been seized with severe abdominal pain and distension, followed by collapse and death in one or two days; and blood has been found in the bowel and in the peritoneal cavity. Very similar results may follow thrombosis of this artery, but the symptoms are more slowly developed.

The effects of embolism and thrombosis of the vessels of the *liver* are described under Pylephlebitis.

In the *pulmonary* circulation, embolism of the largest trunks may occur, commonly as a sequel to femoral thrombosis, when death is often quite sudden. At other times, the event is signalised by sudden collapse, sense of suffocation, and urgent dyspnœa; from which, though generally fatal, cases of recovery have been recorded. Pulmonary infarcts are mostly the result of embolism of the smaller vessels. They are the conical hæmorrhagic masses which are seen in the lungs in chronic heart disease (*see* p. 592). Their occurrence often gives rise to hæmoptysis, and if they are large, there may be dulness and deficient respiratory murmur at the surface of the chest corresponding to them. Pyæmic infarcts of the lung have been already described (*see* p. 162).

Fat-embolism of the pulmonary capillaries is a rare result of injuries, which may allow the passage of fat into the vessels. The symptoms are dyspnœa, prostration, red frothy sputum, quick pulse, cyanosis, and râles over the lungs.

Embolism of particles of *new growth* is no doubt the cause of fresh growths in remote parts.

Treatment.—This is chiefly palliative. The pain of embolism may be relieved by local anodyne applications; if the large artery of a limb is obstructed, the limb should be wrapped in cotton wool, or oiled lint; and surgical measures may have to be considered. Citric acid has recently been credited with solvent properties, and it may be given internally in doses of 15 or 20 grains for the resolution of thrombosis.

FUNCTIONAL DISORDERS OF VESSELS

EXCESSIVE PULSATION, OR THROBBING, OF ARTERIES

This occurs normally after exercise; abnormally in fevers, aortic disease, and some so-called nervous conditions. In exercise the main cause is, no doubt, the excessive force of the cardiac pulsations; in aortic disease the hypertrophied heart, and in fevers the excited heart, must have a large share in the production of the symptom. In other cases the undue pulsation is explained by the condition of the muscular elements contained in the tunica media of the artery wall. Upon these muscular elements depends the tone of the vessel. If they are unusually relaxed the tone is lowered (*hypotonus*): if they are unduly contracted the tone is high (*hypertonus*). In the former case, hypotonus, the artery wall yields to the force of the blood wave, and throbbing or excessive pulsation occurs. In exceptional cases this may be felt all over the body; more often it is limited to a small area. One of the familiar examples is the following:

Excessive Pulsation of the Abdominal Aorta.—This is often mistaken for abdominal aneurysm. It is more frequent in women than men, and occurs between the ages of twenty and forty-five. The patients are generally nervous, hysterical, or hypochondriacal; and it is often associated with some dyspeptic symptoms. There is constant complaint of pain, distress, and throbbing over the abdominal aorta, which can be felt and seen beating with unusual force. If it be carefully examined, it will be found that its outline is cylindrical, like that of the normal vessel, and that there is no fusiform or saccular enlargement. Firm pressure with the stethoscope may elicit a slight murmur, but usually there is no more than the dull sound of impact of the vessel against the instrument. The rest of the circulatory system is normal. The trouble may continue for months or years, without any material alteration.

Treatment.—Gastric disorders should be met by appropriate methods, and the bowels should be kept open. Exercise, fresh air, the avoidance of sedentary occupations, or of the chance of brooding over the complaint, should be enjoined; and, medicinally, bromide of potassium in full doses should be tried.

RAYNAUD'S DISEASE

This disorder, first described by M. Raynaud in 1862, as local asphyxia and symmetrical gangrene of the extremities, is due to a spasmodic contraction of the arterioles, whereby the circulation in the affected parts is retarded, so as to cause a temporary "deadness" or lividity of the part, or is obstructed to such an extent or for such a long period as to be followed by actual gangrene.

Ætiology.—It is much more frequent in women than in men, and is first noticed commonly between the ages of fifteen and thirty, or even in childhood. Many patients are delicate, or anæmic, nervous or hysterical, but some seem to have been in good health until the occurrence of the disease. Hæmoglobinuria, peripheral neuritis, various skin eruptions, and, rarely, ague, have occurred in association with Raynaud's disease. Cold and emotional disturbance are exciting causes.

Symptoms.—Raynaud describes these as occurring in three degrees of severity. The simplest and least severe is one of *local syncope*, in which spontaneously, or from cold or mental emotion, one or more fingers turn white, cold, numb, and insensible to touch. The condition lasts from a few minutes to several hours, and recovery is accompanied by a good deal of pain.

In the second degree, *local asphyxia*, the fingers are more or less cyanosed; they are bluish-white, violet, slate-coloured, or even black. Pressure upon them produces a white spot, which only slowly regains the former livid colour. The adjacent part of the extremity is often slightly swollen, and there is a livid marbling of the limb for some distance above it. There are, with this, always much pain, and complete anæsthesia. Recovery is accompanied by tingling and pricking; and the livid tint gradually passes through scarlet to the natural pink colour.

The third degree is the condition known as *symmetrical gangrene*. Sometimes this begins with pallor of the fingers, which then become lilac, and afterwards violet, with acute pain, tingling, and sensation of burning heat, though the finger ultimately becomes quite cold to the touch. In other cases the finger is at first livid red, with itching and tingling, and finally is the seat of severe pains. Then in either case there is livid mottling of the adjacent limb, and the fingers become black and insensible to touch; vesicles or bullæ containing sero-purulent fluid form on them, and burst, leaving small ulcers, which shortly heal, while the lividity gradually subsides. With a repetition of this process, numerous small cicatrices may form on the affected part, and the fingers acquire a shrivelled, pinched, parchment-like aspect. The skin may desquamate, and the nails may fall off. In other cases, without the formation of bullæ or phlyctenulæ, the fingers and toes become black, shrivelled, and gangrenous; and then a superficial layer of skin, or even some portion of the deeper tissues, separates as a slough in the course of a few weeks. The most marked symptom accompanying these severe cases is intense pain, of paroxysmal character, radiating to other limbs; the pulse may be thin or compressible, but is always perceptible, and the general health of the patient may be remarkably little affected. The toes are attacked as well as the fingers, and sometimes before them; and the nose and ears may be livid, but do not often slough.

The attacks occur at intervals of weeks or months, and in some

cases, after repeated slight attacks, the fingers remain in a permanently benumbed or shrivelled condition.

Diagnosis.—*Senile gangrene* is distinguished by the age of the patient, by the gangrene affecting a single limb, and generally a lower limb, by its progressive course, and by the diseased condition of the artery of the limb. *Chilblains* present a certain resemblance to local asphyxia, and perhaps may have an allied pathology; they occur from definite exposure to cold.

Prognosis.—Many cases recover. Death is rare as a direct result of the gangrene.

Treatment.—A strong continuous galvanic current should be tried, with the anode at the back of the neck, and the kathode over the sacrum and lower lumbar region; or the affected limb should be immersed in a basin of salt and water, in which one electrode of the battery is placed, while the other is applied to the top of the limb. Shampooing the limb and diffusible stimulants internally may also be employed. Cold and excitement should be avoided.

INTERMITTENT CLAUDICATION

Intermittent limping or *claudication* may be considered under functional disorders of vessels, although in most cases there is an underlying structural change. In this rare condition the patient finds that after walking a certain distance he has weakness in one or other leg, with stiffness, heaviness, numbness, pricking sensations, pains and cramp, so that he necessarily limps in his gait. The painful sensations increase as he progresses, and he is at last obliged to stop. The foot or leg shows signs of circulatory disturbance; it becomes red or mottled, and swollen; and the toes may be white and "dead." After a period of rest, the symptoms gradually subside. In the majority of cases there is evidence of sclerosis of arteries or veins, or of obliterative arteritis; and in nearly all there is an absence of pulse in the dorsalis pedis artery, or in the posterior tibial of the affected limb. It is, accordingly, a disease of adult life; and gout and syphilis, and indulgence in tea, tobacco, or alcohol, are often among the antecedents. It is more common in men than in women.

In some cases slight muscular wasting and degeneration of the peripheral nerves (peripheral neuritis) have been observed. In many instances the complaint has resulted in dry gangrene of the limb; and it has been associated in a few cases with Raynaud's disease of the upper extremities.

In such cases as do not present any evidence of arterio-sclerosis or obliterative arteritis, it is assumed that the condition is due to arterial spasm.

The prognosis is bad, but the attacks may go on for years.

Treatment.—The patient must limit his exercise, and avoid quickening the circulation up to the point at which the obstruction in the vessel will begin to operate. Frequent rests in bed

may be advisable. Iodide of potassium may be tried in syphilitic cases; and the local remedies used in Raynaud's disease may be employed here also, the constant current, electric baths, warm baths, high-frequency currents, and gentle massage.

If gangrene occurs, the pain may be relieved by morphia, and the part should be amputated at a suitable opportunity.

ACROPATHY

This name has been used to describe a number of affections distinguished by sensory, vasomotor, or trophic changes situated especially in the extremities. Some of them are, no doubt, related to Raynaud's disease, but their pathology is obscure.

Erythromelalgia.—In this condition, first described by Weir Mitchell, there are attacks of acute pain in the feet and legs associated with, or followed by, dilatation of the blood-vessels, the part becoming bright red, or deep purple in colour, with shiny surface, prominent veins, and perhaps sweating. The pain is acute, burning and throbbing. The attacks are brought on and aggravated by heat, exercise, and a dependent position of the limbs; and some relief is obtained by cold and by elevation of the limbs. The attacks last at first a few hours; but with the progress of time they are more persistent, and perhaps at the same time less severe. It occurs in men of early middle age, and rarely in children.

Two of Mitchell's cases subsequently developed spinal symptoms, and other cases have been seen to be associated with tabes, syringomyelia, and disseminated sclerosis. Some cases of erythromelalgia appear to be due to ergotism, and an analogous condition has been observed in the arsenical poisoning of beer-drinkers.

The treatment is mainly symptomatic; by cold, suitable position, and the use of morphia. Faradism and massage have also been of use.

Acroparæsthesia.—In this there are disagreeable or painful sensations, tingling or numbness, or "pins and needles" in the hands and feet. It may be accompanied by vasomotor disturbances. It is more common in women than in men. The disagreeable sensations are felt usually in one or both arms when the patient wakes in the morning, and after a time the symptoms subside. Sometimes the hand is paler, or redder than normal, or even swollen. In a few cases there appears to be a sufficient cause in much use of the hands in some occupation during the day, or in a faulty position of the arms during sleep; but often no cause can be discovered. The pathology is not clear: it has been attributed to vasomotor spasm, but in many cases there is no evidence of this. It has been seen in general paralysis, tabes dorsalis, and allied disorders; but as a rule it is independent both of these, and of hysteria. The treatment consists of rest, tonics, potassium bromide at night, and the constant current.

Sclerodactylia.—This is a trophic change in the skin of the fingers,

by which it becomes shrunken, atrophied, and glossy. The fingers are deformed, the nails fall, and ulcerations occur.

Acrocyanosis is also described, consisting of a violet tint of the hands and feet, accompanied by slight pains.

ANGEIO-NEUROTIC ŒDEMA

This is another curious affection apparently connected with the vasomotor apparatus. Circumscribed swellings appear in various parts of the body, for instance on the face, the eyelids, the hands or legs, in the throat or in the tongue. They are not inflammatory, and not dependent upon gravity ; they are not painful, but may be accompanied by burning, pricking, and itching. They appear suddenly, last from two to six hours or more, and recur frequently, even daily ; on the skin they are generally harmless, but œdema of the larynx has frequently proved fatal. Gastro-intestinal symptoms are usually present, such as colic, nausea, and vomiting, and are attributable to an acute œdema of the gastric or intestinal mucous membrane. The disease is often hereditary, occurring in several members of the same family, in two or three generations. Little is known as to its cause. Quincke suggested an intestinal auto-intoxication.

Treatment has but little effect; bromides and calcium chloride have failed. Quinine, nitro-glycerine and thyroid extract have given relief in a few cases. Laryngeal œdema may require intubation or tracheotomy.

DISEASES OF THE MEDIASTINUM

MEDIASTINITIS

Inflammation of the mediastinum may be suppurative or non-suppurative. The former, or *mediastinal abscess*, arises from numerous causes, of which injuries by bullet, stab, or blow, and tuberculosis of the lymphatic glands are the most frequent, while occasionally it follows upon pneumonia, pleurisy, erysipelas, or enteric fever. The abscess may be in the anterior or in the posterior mediastinum, more often in the former. The chief symptoms are sternal pain and pyrexia. Physical signs will only be apparent if the abscess reaches a sufficient size, when there may be dulness, localised tenderness, œdema over the sternum, and ultimately fluctuation at the border of that bone. The pus must be evacuated as soon as possible, and, if necessary, the sternum must be trephined or a portion resected.

Adhesive, simple, or non-suppurative mediastinitis has been less often recognised than the suppurative form. It may also arise from traumatism and general infectious diseases, but its most common associations are pleurisy and pericarditis, especially the

latter, forming then a *mediastino-pericarditis*, or *mediastinitis fibrosa*. In these circumstances the tissues of the anterior mediastinum may be matted together to form dense fibrous tissue, and with this there are pericardial adhesion or thickening, sometimes pleural adhesion or fluid, and often ascites, with or without chronic peritonitis. The physical signs are those enumerated under Adherent Pericardium (*see* p. 635). The symptoms are dyspnœa, dropsy of the legs, enlarged liver and ascites, albumin in the urine, and sometimes the pulsus paradoxus. When the mediastinitis is pronounced, there may be cyanosis and œdema of the neck, face, and arms. The cases thus present the appearance of heart disease without cardiac murmurs; or of cirrhosis of the liver with pleural complications, if the abdominal distension is out of proportion to the œdema of the legs; or of mediastinal tumour if cyanosis and œdema of the upper part of the body are marked. Apart from these last, most of the symptoms can be explained by the cardiac failure consequent on adherent pericardium. The physical signs on the side of the heart are often very few, but if adherent pericardium can be diagnosed on its own merits, the evidence of past or present pleurisy makes mediastinitis very probable. At the same time its importance seems somewhat small by the side of the immediate cardiac lesions.

Treatment.—This resembles the treatment of valvular disease; purgatives and diuretics will have to be employed, and the ascites often requires paracentesis.

The operation of *cardiolysis* has given much relief in some cases. This consists in resection of the left fourth, fifth, and sixth ribs, perhaps also the third and the seventh, with their costal cartilages, from the front of the heart, so as to liberate it in part from the tissues which hamper its action

TUBERCULOSIS OF THE BRONCHIAL GLANDS

(*Bronchial Phthisis*)

Ætiology and Pathology.—The former is similar to that of tubercular infection in general; and the bronchial glands are infected commonly from the lesions of pulmonary phthisis. But there is this occasional point of difference, that in children the glands may caseate, enlarge, and even suppurate with very little or no evidence that the lungs are involved; and these are especially the cases to which the name of bronchial phthisis has been given. The bronchial glands go through the same changes as other organs when invaded by tubercle. Gray granules are at first formed, and caseation follows; the glands become enlarged, and may suppurate or become calcareous. As a result of their enlargement they press upon surrounding parts, especially the œsophagus and the trachea or main bronchi. If they suppurate they may discharge into one of these passages, or they may lead to abscess in the mediastinum.

Symptoms.—Enlarged bronchial glands constitute a mediastinal tumour. They cause pain in the chest, cough, and dyspnœa, and perhaps slight swelling of the face and neck, dysphagia, and hæmoptysis. The cough is of most interest. It is sometimes harsh and altered in quality, as if the larynx were involved; and this is probably from some pressure of the glands on the recurrent laryngeal nerves, producing paresis of one or both cords. The cough may be constantly hacking, or spasmodic like that of pertussis. If the pressure on the trachea is considerable, there is severe dyspnœa with stridor, and impending suffocation; and this is especially likely to be the case if the glands are suppurating and rupture is imminent. When rupture takes place, pus is expectorated, and there is some danger of asphyxia in young children. Occasionally a detached fragment of gland has been impacted in the respiratory passages, and has caused death. The puffy swelling of the face and neck is due to the pressure on the superior vena cava or one or other innominate vein. There is more or less pyrexia, according to the stage and the rapidity of the caseating process.

Physical Signs.—If the granular mass is of great size, it will cause dulness between the scapula and the spine on one or both sides; and more rarely over the upper part of the sternum. The auscultatory signs are variable, even independent of any co-existing phthisis. There may be deficient breath-sounds over the lung on one or other side; or bronchial breathing and bronchophony in the neighbourhood of the enlarged glands. A venous murmur is sometimes heard over the manubrium sterni, especially when the head is thrown back, and this has been attributed to the pressure of the glands on the left innominate vein. But such a murmur was heard in 13 per cent. of children with no evidence of enlarged glands, and it is probably only the result of transmission of a jugular murmur (J. E. H. Sawyer), which is itself very common in quite healthy children.

Diagnosis.—The symptoms and physical signs of what is practically a mediastinal tumour in a young child, with a family history or other evidence of tubercle, will generally enable the disease to be recognised; but in a small child it may be easily confounded with empyema, until tested by exploration or by Röntgen rays.

The **Prognosis** is not absolutely unfavourable. Health may be restored completely after the discharge of pus; and other cases, no doubt, get well on calcification of the tubercle.

Treatment.—This must be conducted on the same principles as that of tubercle in general. Good food, fresh air, and tonics—such as the syrup of iron iodide or of iron phosphate, Parrish's food, or cod-liver oil—are required. Tuberculin may be used both for diagnosis and treatment. Local stimulants, such as iodine, may also be useful, and cough and pain must be met by small doses of anodynes, Dover's powder, compound tincture of camphor, or syrup of poppies.

MEDIASTINAL NEW GROWTHS

New growths in this situation arise commonly from the bronchial or posterior mediastinal lymph-glands, but it may be from the thymus, and possibly from the fatty or subserous tissues, or the periosteum. The most frequent form of new growth is a lymphoma, or lympho-sarcoma; spindle-cell sarcoma, carcinoma, gummata, and rarely dermoid cysts also occur. Inflammatory infiltration and abscess also produce many of the effects of definite tumours. In the mediastinum these tumours are adjacent to the great vessels, the trachea, the roots of the lungs, and the œsophagus. They may extend above the clavicle into the neck.

Symptoms.—These are, for the most part, due to the pressure of the new growth upon the important structures in the chest. This results in (1) pain; (2) obstruction of veins, arteries, air-tubes or œsophagus; (3) compression of one or both recurrent laryngeal nerves; (4) displacement of organs; and (5) deformity of the chest.

The *pain* is variable, it may be felt behind the sternum, or in the back, or may radiate in the course of the nerves.

Obstruction of the veins is one of the most important indications of mediastinal tumours, since veins have little power of resisting the growth of tumours, which readily press their walls together, and even grow through them and project into the interior, or lead to thrombosis and fibrous stricture. As a result of obstruction of the superior vena cava, the veins of the head, neck, face, arms, and upper part of the chest are dilated, and if it occurs rapidly these parts become immensely swollen from œdema, contrasting curiously with the lower part of the body. The blood, however, finds its way to the heart, by anastomoses under the skin between branches of the intercostal and the abdominal veins; and these become visible as a close network of small blue tortuous vessels on the chest and large vertical tortuous veins on the abdomen. Even with this compensation the venous current may be much obstructed, and, on stooping down or making any exertion, the face becomes still more congested and cyanosed.

In cases of obstruction of the inferior vena cava a similar communication takes place, but there is a difference, in that the flow of blood on the surface is entirely downwards in obstruction of the superior vena cava, and upwards in obstruction of the inferior cava. This last, however, rarely results from mediastinal tumour, although it is possible for a malignant growth to reach the inferior cava, just above the diaphragm.

Arteries often maintain their course through a tumour unmolested. They are sometimes compressed, with the effect of weakening or obliterating the peripheral pulse.

The results of *compression of the trachea and bronchi* have been already described (pp. 480, 498). If the *œsophagus* is involved

there will be difficulty in swallowing, and an œsophageal bougie will meet with resistance.

The paralyses of the vocal cords, which follow pressure on the *recurrent laryngeal nerves*, are similar to those caused by aneurysm. The left nerve is more exposed than the right from its lower position, and the results will be abductor paralysis, or complete paralysis, according to the degree of compression. Sometimes there are spasmodic attacks of dyspnœa.

The *displacement of organs* and *deformity of the chest* depend on the size and position of the tumour. The heart may be pushed to the right or left, the liver downwards towards the abdomen, or one lung may be compressed against the ribs. Deformity may be quite absent, especially if the tumour arises in the posterior mediastinum. But there may be enlargement of one side from effusion of fluid into the pleura, caused by the growth pressing on the veins in the root of the lung; or retraction of one side if the growth compresses the bronchus. Tumours in the upper part of the chest may extend into the root of the neck, or be accompanied by enlargement of cervical glands in this situation. Dyspnœa, spasmodic cough, expectoration of mucus, hæmoptysis, faintness, attacks of syncope, and palpitation are additional symptoms in many cases. Paraplegia occurs in exceptional cases from invasion of the spine.

The *general condition* of the patient is dependent often upon the extent and duration of the lesion. At first he may show little change, but cachexia and wasting must come in due time. Definite and prolonged *pyrexia* is sometimes present, as, for instance, in the following circumstances: if the growth is lymphadenomatous, there may be the characteristic recurrent pyrexia of Hodgkin's disease; carcinoma is sometimes attended by pyrexia, which is also liable to vary in intensity from week to week; if a bronchus is obstructed so that sepsis occurs within it, pyrexia will be caused.

Physical Signs.—The most important is the decided dulness which a tumour produces if it reaches the surface of the chest, so as to displace the lung, or if it compresses the lung between itself and the chest-wall; but a large tumour may exist in front of the spine without appreciably altering the signs of percussion. The auscultatory sounds will depend on the presence or absence of fluid in the pleura, and on the relation of the tumour to the root of the lung (*see* Aneurysm, p. 651).

Diagnosis.—Mediastinal new growth is most readily confounded with an *aortic aneurysm*, which is itself a mediastinal tumour, occupying the middle or superior mediastinum when it grows from the arch, and the posterior mediastinum when it arises from the thoracic aorta. A diagnosis is, nevertheless, very desirable, since the prognosis as to duration of life and temporary relief of symptoms is much more favourable in the case of aneurysm. Many of the symptoms are necessarily the same; they both press upon and displace important structures, such as the œsophagus, the trachea or bronchi, and the large nerve-trunks. One of the most important distinctions

is the fact that aneurysms implicate arteries, and thus may lead to suppression of pulse on one or other side; while new growths commonly spare the arteries, but compress and project into the veins, producing venous distension and œdema. The presence of a murmur, and the duration of the symptoms for more than twelve or fifteen months, are in favour of aneurysm; as is also the distension of the lung as a result of bronchial stenosis, before the stage of collapse (*see* p. 498). Enlargement of the cervical glands or the existence of tumour in other parts of the body, speaks for new growth; in this connection the mammæ or the testes should always be examined. A diagnosis from venous obstruction alone is unsafe, since thrombosis and fibrous stricture of the vena cava may occur independently of aneurysm, tubercular masses, or new growth. The stridor produced by compression of a bronchus should not be mistaken for the rhonchus of *bronchitis*. The former is constant in time and position; the latter is variable, and changes from place to place within a few hours, or disappears at intervals. Assistance in diagnosis may be obtained from Röntgen rays, the bronchoscope, or the œsophagoscope.

The **Prognosis** is bad, and the duration rarely more than a few months.

Treatment.—This is mainly palliative, and very little can be done beyond relieving pain by opium or morphia. Symptoms threatening life, such as laryngeal asphyxia, must be met as they arise.

DISEASES OF THE ORGANS OF DIGESTION

PHYSICAL EXAMINATION OF THE ABDOMEN

The abdomen is accessible to the same methods of examination as are employed in the case of the lungs and heart—namely, inspection, palpation, mensuration, percussion, and auscultation; and in most instances it is desirable that the patient should be in the recumbent position, with the head rested.

The anatomical regions commonly recognised are in the middle line, the epigastric, the umbilical, and the hypogastric or pubic region; and on either side the hypochondriac below the costal margin, the iliac or inguinal near Poupart's ligament, and between them what has been called the lumbar, but would be much better called the lateral region, or flank. As a fact, in most persons the hypochondriac and iliac regions together leave little room in front for a lateral region. Posteriorly between the twelfth rib and the iliac crest is the lumbar region, properly so called. As in the chest, accurate localisation of a lesion requires measurement from easily recognised parts, like the umbilicus, xiphi-sternum, middle line, pubes, anterior superior iliac spine, or tip of the eleventh rib.

INSPECTION

The first thing to notice is the size of the abdomen. This is extremely variable even within the limits of health. It may be uniformly much enlarged; but it requires the help of other methods of examination to determine whether this is due to a collection of liquid in the peritoneal cavity (*ascites*), to gas in the intestines (*meteorism, tympanites*), to fat in the parietes and omentum, or to some tumour, such as an ovarian cyst. Uniform and symmetrical retraction of the abdomen is seen in starvation, in emaciating diseases, and in death from cerebral diseases, such as tubercular meningitis and intracranial tumour.

By inspection may be observed various local enlargements or prominences, such as result from tumours or enlargements of different organs. Many of the tumours which occur in the abdomen may, under favourable circumstances, be visible on the surface; this is much more likely to occur when the patient is thin, and the abdomen empty, than when the patient is fat, and the abdomen is distended with ascites or flatus. Amongst those more commonly observed are enlargements and tumours of the liver, cancer of the

stomach, and dilated stomach, distended intestines in cases of obstruction, infiltrated omentum and adherent intestines in tubercular peritonitis, enlarged spleen, hydronephrosis, the pregnant uterus, ovarian and other cysts, and a distended bladder. Local enlargements in the upper part of the abdomen may cause asymmetry of the thorax by driving upwards and outwards the lower ribs on one side, thus enlarging the angle between the costal margin and the middle line. This is especially seen in cases of hydatid, cancer, and abscess of the liver.

It is important to note the relation of the abdomen to the respiratory movements already referred to in connection with the chest (*see* p. 446); the descent of the diaphragm is impeded by much distension, and is checked by acute inflammation of the peritoneum, so that in these cases respiration is almost entirely thoracic. In other cases respiration affects materially the position of the organs in immediate contact with the diaphragm—namely, the liver, spleen, stomach, and kidneys; whereas, organs or tumours situated lower down, or connected with the posterior wall of the abdomen, are much less influenced by the descent of this muscle. The pulsations of the aorta, or of the right ventricle, or of an aneurysm, or of the enlarged liver in tricuspid regurgitation, and the peristaltic movements of a dilated stomach or of the intestines, may sometimes be seen, the latter being visible in proportion to the thinness of the abdominal parietes, and the vigour of the peristalsis.

The sudden expiratory effort of coughing will sometimes produce local prominences, which are not present in ordinary circumstances. These are always due to weakness or deficiencies of the abdominal wall, and the projection constitutes for the time being a hernia. Besides the familiar inguinal and umbilical herniæ, it is common to see a protrusion in the middle line from yielding of the parietes between the two recti muscles; and occasionally the abdominal walls, just above Poupart's ligaments, bulge in coughing. A similar localised bulging in other parts may result from a limited paralysis of the abdominal muscles following upon neuritis.

Röntgen Rays.—Under inspection again we must consider this method of examination, which has proved to be of great value in the case of the stomach and intestines, by giving shadows of their contents when these are of a metallic nature. If a solid salt of bismuth in some quantity is taken internally, its course through the œsophagus, stomach, and intestines can be closely watched on the screen, and important information is thus obtained as to the size and position of the hollow viscera, and as to the rate of transit of the bismuth contents.

PALPATION

This is undoubtedly the most important means of examining the condition of the abdomen. The abdominal walls should be relaxed as much as possible, and for this purpose the patient should be in the recumbent or semi-recumbent position, and the head should be

supported, for if the patient raises his head—*e.g.*, to see what is going on—the recti abdominis become tense. The relaxation of the abdominal walls is sometimes assisted by raising the patient's legs, but they must be supported in that position by a pillow under the knees. This procedure compels the observer to examine very much from one side, rather hampers the use of the hands, and can often be dispensed with.

In many patients the abdominal muscles are persistently tense, and the abdomen is palpated with difficulty. In such cases the patient should be asked to breathe deeply in and out, and should be engaged in conversation while the hand is on the abdomen; or he may be asked to lift up his head from the pillow, and keep it raised for about a minute, when the muscles will become exhausted, and for a moment afterwards the abdomen may be lax enough for the purpose. If these means fail, and an examination is of the first importance, chloroform should be administered.

The examination of the abdomen should be made with great gentleness; the hands should be warm, and should be laid flat upon the surface, and care should be taken not to force the finger-tips suddenly into the abdomen, whereby the muscles are made to contract, and trustworthy results are impossible. During the movements of deep breathing, enlargements of organs or new growths, especially in the upper part of the abdomen, may be detected, which would otherwise perhaps escape recognition. When examining the sides of the abdomen, or the flanks, the observer should never neglect to employ the *bimanual* method—that is, one hand should be placed under the twelfth rib, and another on the abdomen in front; if one hand be pressed towards the other which is still, the slightest enlargement or resistance can generally be appreciated. In particular cases it may be desirable to examine the patient in the knee-elbow position.

In the normal abdomen there is scarcely any resistance to the movement of the hand in all directions. The solid organs, liver, spleen, and kidneys are almost entirely within the bony thorax; the left lobe of the liver, which lies across the epigastrium, is of small bulk, thin, and soft; the hollow viscera yield readily to the hand, and often nothing can be recognised, except, in thin people, the pulsation of the aorta or iliac vessels.

By palpation in disease we can recognise changes in shape or size of the organs, and the existence of tumours, and can obtain information on the following points:

The condition of tenseness or relaxation of the abdominal wall, which may be local or general.

The presence of tenderness, general or local: this may be elicited directly the hand touches the abdomen, or only when deep pressure is made.

Various kinds of movement may be felt in the abdomen—the pulsations of the normal vessels, or of an aneurysm, or of the liver in heart disease; the peristaltic movement of the bowel; the

movements of air in the intestine (borborygmi); the crackling or gurgling due to air and liquid, which may be felt sometimes on gentle pressure over the cæcum in enteric fever; the coarser movements or splashing of air and liquid in a dilated stomach when somewhat sudden pressure is made upon it; and the friction of inflamed peritoneal surfaces.

Under palpation also must be included two of the methods by which ascites or fluid in the peritoneal cavity may be recognised—namely, *fluctuation* and *displacement* (*see* Ascites).

MENSURATION

By measurements we may watch and record the progress of enlargements of the abdomen from ascites or tumour, and the hepatic or splenic dulness may be exactly estimated (*see* Percussion). Sometimes mensuration may help the diagnosis of ascites from ovarian disease, by comparison of the distances from the umbilicus to the ensiform cartilage, to the pubes, and to the iliac crest (*see* Ascites); and it will confirm the evidence of inspection with regard to hepatic tumours displacing the ribs.

PERCUSSION

It is in reference to percussion especially that we must remember that the abdominal cavity extends up into the lower parts of the bony thorax. In health the abdomen is resonant over so much of the combined surfaces as corresponds to the intestines and to the stomach—that is, all the parts below the ribs, and the costal cartilages and lower ends of the ribs on the left side below the heart. It is dull over the parts which correspond to the liver and spleen—that is, for the liver, the ribs of the right side below the upper border of the sixth in front, and the eighth at the side, and for the spleen the ninth, tenth, and eleventh ribs on the left side just behind the anterior axillary line; and with very light percussion one may recognise the extension of the left lobe of the liver across the epigastrium. The relative areas of dulness and resonance may be much altered by changes in the amount of gas in the hollow viscera, and the dull areas of the liver and spleen are moved downwards in inspiration and upwards in expiration. There is, further, much difference in the quality of the percussion note over the stomach and different parts of the intestine.

Alterations in the size of the liver and spleen, or the existence of solid tumours or cysts, will give rise to new areas of dulness, and such dulness will, as a rule, be accompanied by resistance appreciable by palpation. As constant reference to these altered conditions will be made under the diseases of the different organs, it is not necessary to specify them here.

In percussion also we have another valuable method of recognising ascites, since liquid in the peritoneal cavity, unless retained

by adhesions, tends always to be lower than the intestine, which contains gas; and if the patient is examined as he lies on either side and on his back in succession, the dull and resonant areas will be found to alter their positions accordingly (*see* Ascites).

AUSCULTATION

Friction sounds are occasionally heard over the liver and elsewhere in peritonitis; abdominal aneurysms may be accompanied by murmurs. By combining auscultation with percussion (auscultatory percussion) the differences in the sounds produced over the hollow viscera may be recognised, which will allow their limits to be more accurately mapped out. Combined with palpation, auscultation helps us to recognise the splashing sounds in a dilated stomach; but they can be often heard without the stethoscope.

DISEASES OF THE MOUTH, TONSILS, AND PHARYNX

STOMATITIS

Inflammation of the mouth, or *stomatitis*, occurs as a general catarrhal condition involving the cheeks, gums, tongue and lips; and in more localised forms, as aphthous, ulcerative, and gangrenous stomatitis, which are almost certainly due to micro-organisms. At the same time it is clear that some special conditions are required for the operation of micro-organisms, since the mouth of healthy persons contains innumerable micro-organisms, among which are staphylococci, streptococci, torulæ, and sometimes pneumococci, and diphtheria bacilli. The lesions of some skin diseases also sometimes involve the buccal mucous membrane, such as those of herpes, pemphigus, and urticaria. Stomatitis limited to the gums is called *gingivitis*. Different forms of gingivitis are seen in scurvy, and in acute leuchæmia; and as local results of dental diseases in the form of *pyorrhœa alveolaris* and *gumboil*.

It is necessary to lay especial stress upon the importance of a healthy condition of the teeth in relation, not only to stomatitis, but to conditions of general ill-health. Carious teeth, diseased stumps, accretions of tartar upon the teeth, and accumulations of food particles, favour the growth of many kinds of septic organisms; pus is formed in pockets by the side of the teeth, and thus not only is constantly taken into the stomach, but may cause local infections like angina ludovici, and supplies toxins which by their absorption lead to severe forms of anæmia, deficient resisting power, and other conditions of ill-health attributable to auto-intoxication. This condition is conveniently known as *oral sepsis*.

CATARRHAL STOMATITIS

Ætiology.—Catarrhal stomatitis may be set up by chemical or mechanical irritation, such as contact with acids or alkalies, excessive drinking, or the presence of broken or carious teeth; secondly, by inflammation spreading from adjacent parts, such as the nose or naso-pharynx; thirdly, by the action of some poisons, viz., mercury, lead, and arsenic; and fourthly in consequence of some general and mostly infectious conditions, such as measles, variola, syphilis, scurvy, leuchæmia, and others.

Symptoms.—These are swelling and increased redness of the mucous membranes of the gums, lips, and cheeks, swelling of the tongue, salivation and increased secretion of buccal mucus, which adheres as a coating to the surface, and swelling of the neighbouring lymphatic glands. Mastication and deglutition are painful, and the breath may be offensive. In later stages abrasion and superficial ulceration take place.

Treatment.—All causes of irritation should be as far as possible removed: and antiseptic washes should be employed: such as boric acid (2 to 5 per cent.); potassium chlorate (3 per cent.). In later stages much more astringent solutions, such as alum (5 grains to the ounce) or glycerine of tannin.

APHTHOUS STOMATITIS

This occurs in children, especially about the time of the first dentition, and less frequently in adults: it consists in the formation of circular gray patches, or aphthæ, on the gums, tongue, and the inside of the lips and cheeks. They are from 3 to 5 mm. in diameter, slightly raised above the surface, and, though looking like vesicles, are really caused by a fibrinous exudation beneath the epithelium. After a time the epithelium is shed, and small ash-gray ulcers with narrow red margins are left. Children thus affected are restless and feverish; there is slight salivation; and sucking or mastication is painful. The ulcers commonly heal in a few days, but may recur frequently in some patients. In adults the aphthæ are rarely so numerous as in children.

Treatment.—Chlorate of potassium should be given internally (2 or 3 grain doses for a child), combined with the local use of antiseptic solutions; such as those of borax (15 grains to water ℥j); glycerinum boracis, boric acid (1 per cent.), or carbolic acid (1 per cent.). The application of nitrate of silver in adults at once relieves pain and often quickly cures.

ULCERATIVE STOMATITIS

Ulcerative stomatitis is more common between the second dentition and puberty, but is seen at other periods of life. It has occurred in an epidemic form amongst soldiers in camp and prisoners in gaol; and it is probably due to micro-organisms, but none has certainly been identified as specific. It attacks especially those who are in ill-health or badly nourished. It begins at the free margins of the gums, which become red, swollen, detached from the teeth, and may bleed on slight pressure, or in the movements of mastication. Ulceration then takes place, the ulcers being often deep, ragged, covered with a gray or yellowish purulent coating, and surrounded by a thin red margin. The process spreads irregularly over the gums, and involves the lips and cheeks as well. Sometimes the ulceration extends down to the periosteum, and superficial necrosis of the jaw results. The teeth are loosened, there is free salivation, and the cervical lymphatic glands are enlarged and tender. The tongue and palate are inflamed at the same time, but are not generally ulcerated. The constitutional symptoms are often rather severe, and pyrexia is present. Mastication and swallowing are of course, painful and difficult.

The **Prognosis** is favourable, recovery commonly taking place in one or two weeks.

Treatment.—The best results are obtained from the internal use of chlorate of potassium, which should be given in doses of from 5 to 15 grains, three times a day, according to the age of the patient; and the mouth may be frequently washed with solutions of the same salt (10 or 15 grains to water, ℥j), or carbolic acid (5 grains to ℥j), or listerine and water (equal parts). The patient should be supported by good fluid nourishment at the same time

GANGRENOUS STOMATITIS

This disease, also called *cancrum oris* and *noma*, occurs in debilitated children, or those subject to bad hygienic conditions, or those who are recovering from infectious disease, of which measles and enteric fever are most common. It is due to bacterial infection. The changes are very rapid: a small spot of induration appears on the inner side of the cheek, and soon the whole thickness of the cheek is hardened, black in the centre, and reddened around, or in other words, a slough has formed. If it goes on the cheek will be perforated, or if it is on the lips, the gum will be invaded, and the teeth will fall out. There is very little pain or fever, but the child rapidly becomes exhausted and dies.

Treatment.—The only means of saving the child is the prompt destruction of the part by nitric acid, or its removal by the knife. In addition the child must be supported by food and stimulants.

THRUSH

Thrush is seen in weak and badly nourished infants, especially in those who are being fed by hand, or are suffering from diarrhœa; and also in adults, in the last stages of exhausting diseases, such as phthisis, cancer, and enteric fever. Upon the mucous membrane of the lips, cheeks, gums, palate, and tongue, milk-white patches occur, which are irregular in shape, scattered or confluent, slightly raised above the surface, and surrounded by a thin red line. If the patch is stripped off, the mucous membrane beneath is bright red, or even bleeds slightly, and the patch may form again in a short time. It consists of epithelial scales, fat globules, and the spores and mycelium of a fungus which has received various names: *Oïdium albicans, Saccharomyces albicans, Mycoderma vini,* and *Monilia candida.* The fungus develops first in the middle layers of the epithelium, and spreads thence in both directions to the more superficial and the deeper layers. It is probable that the growth of the fungus is the cause of the stomatitis which accompanies it; but it is stated by Vogel that the deposit is favoured by the secretions of the mouth, which are acid before any white patches appear. Children who have thrush and diarrhœa frequently have excoriations about the anus, which lead to the popular notion that the thrush has "gone through" the child; but though in severe cases thrush may extend to the pharynx and œsophagus, it does not occur on parts covered with cylindrical epithelium. The anal rash is either erythema intertrigo or a congenital syphilide. A certain amount of local discomfort, with pain on swallowing or sucking, results from thrush, but symptoms beyond these are chiefly due to the condition of health preceding it.

Treatment.—We should aim at improving the general condition of the patient. In infants the food must be rendered suitable, and the diarrhœa checked. After every meal the mouth should be carefully wiped out with a fresh piece of soft linen; and the patches should be touched with a solution of borax (10 grains to ℥j) or a little glycerine of borax should be left in the mouth.

ANGINA LUDOVICI

Ludwig's angina, or *submaxillary cellulitis,* is a rare form of severe and often fatal phlegmonous inflammation of the floor of the mouth, and upper part and front of the neck. It is no doubt due to infection from some part of the buccal cavity. Streptococci and staphylococci have been found in different cases. Its treatment is mainly surgical.

CATARRHAL SORE THROAT

Ætiology.—The causes are cold, and exposure to impure air, whether in badly ventilated rooms, in hospital wards, or from open drains.

Symptoms.—In its mildest form there is only some discomfort on swallowing, and nothing may be visible in the throat. In other cases, the soft palate, uvula, pharynx, and tonsils are redder than natural, the uvula is elongated, the soft palate is flaccid, and the pharyngeal veins are dilated. In more severe cases (*ulcerated sore throat*) superficial abrasions occur on the tonsils, palate, and pharynx, the tongue is furred, and there is marked constitutional disturbance. Sometimes there is excess of saliva and buccal mucus; at others there is unusual dryness of the mouth. Talking, as well as swallowing, may be painful.

Treatment.—For this we may use iron, quinine, or other tonics internally, and apply glycerine of tannic acid locally, or give rhatany lozenges. Where it is due to a hospital atmosphere, or other impure air, removal to the country for a few days may be desirable, and in other cases the improvement of the air-supply must not be neglected.

TONSILLITIS

Inflammation of the tonsils occurs in diphtheria, scarlatina, syphilis, and rheumatism, as already described. In addition, we recognise a suppurative tonsillitis and follicular tonsillitis independent of general infection. Streptococci and staphylococci are the organisms chiefly concerned (*see also* Catarrhal Sore Throat *and* Chronic Pharyngitis).

SUPPURATIVE TONSILLITIS

(*Quinsy*)

Ætiology.—This is most common between the ages of fifteen and twenty-five; some persons are very liable to it, and have it repeatedly. Both this and follicular tonsillitis are regarded by some as being part of acute rheumatism, even when occurring quite independently of the articular lesions.

Symptoms.—It may affect one or both tonsils. The tonsil becomes red and swollen to twice its natural size, projecting to the middle line, and pushing the uvula aside; if both tonsils are affected they may meet in the middle line, driving the uvula forwards; the swelling and redness extend to the base of the soft palate. The surface is generally smooth, shining, and deep red or purple in colour. Externally, there is obvious swelling behind the angle of the jaw. The illness often commences with a rigor, and sickness; and the constitutional disturbance is considerable. The

tongue is thickly furred, appetite is lost, and the temperature rises to 103° or 104°. Swallowing and talking are excessively painful, and saliva and mucous secretion collect in the mouth, and require to be constantly expectorated. In from two to four days suppuration occurs; the tumour, which was at first hard, is now softer, and yields to the finger; or the presence of pus may be detected by placing one finger on the tonsil, and another outside behind the angle of the jaw. If left alone, the abscess bursts into the throat, the temperature falls, and recovery quickly takes place in from four to seven days, though convalescence may be protracted for some time longer. Rarely the abscess has burrowed into the neck or chest, or eroded the carotid artery, or caused suffocation by discharging its pus into the larynx.

Diagnosis.—Quinsy may resemble *follicular tonsillitis;* it is more often unilateral, the fever is more severe, the redness extends to adjacent parts, secretion does not accumulate in the follicles, and pus may be eventually detected. Sometimes the two occur together.

Treatment.—In the early stages, ice often relieves the pain; it should be sucked as well as applied to the throat externally. Salicylate of sodium in 10 to 15 grain doses every three or four hours may be given internally, and guaiacum lozenges (3 grains in each) may be sucked every two hours, to diminish the acute symptoms. If suppuration has commenced, hot fomentations and poultices probably hasten it. When pus is detected, an incision should be made into the tonsil with a bistoury, covered up to the last half-inch with plaster, so as to protect the other parts of the mouth. The patient is generally confined to bed, and can take only fluid food. This should consist of milk, beef-tea, and strong broths; and stimulants are usually required. In later stages, quinine and iron may be given in full doses.

FOLLICULAR TONSILLITIS

Ætiology.—This form of tonsillitis also recurs frequently in the same people, and often appears to arise from impure air.

Symptoms.—The tonsil is red and swollen, and presents several yellow prominent spots, which are follicles distended with secretion; and the surface is covered with more or less mucus. The swelling can be felt externally behind the angle of the jaw. In severer forms the secretion of the follicles is more abundant, and they are distended with large bright white plugs, which may present a close resemblance to the white material of diphtheria. Both tonsils are frequently affected. There is moderate constitutional disturbance, furred tongue, slight pyrexia, feeling of malaise, and the same local discomfort as in other forms of tonsillitis. Recovery always takes place.

Diagnosis.—The occasional resemblance to *diphtheria* is most important. Generally, the obvious formation of the plug of secretion within the follicle of the tonsil, or the existence of several on

each side, serves to distinguish them. A single white patch of some extent, apparently only on the surface, is in favour of diphtheria, and an extension to the soft palate is conclusive. The bacteriological test of cultivation for the Klebs-Loeffler bacillus should be employed in doubtful cases.

Treatment should be nourishing and stimulating; internally quinine and perchloride of iron, and port wine in weakly individuals. The tonsils may be painted with astringent or antiseptic solutions, such as glycerine of tannic acid, tincture of perchloride of iron in glycerine (5 drops to ʒj), liquor sodæ chlorinatæ, chinosol (1 in 500 of water), formalin (2 per cent. in glycerine), solutions of alum, or boric acid. Lozenges of potassium chlorate or rhatany, and formamint tablets may also be sucked.

VINCENT'S ANGINA

This is a form of tonsillitis which also presents some resemblances to diphtheria, in that an exudation forms on the tonsils, the cervical glands are swollen, and there are difficulty of swallowing and some fever. A white membrane is seen on the first day, and beneath it ulceration takes place; if the membrane is detached, it forms again, and may become gray, yellowish-gray, or faintly green. Ulceration continues during four or five days, and is sometimes followed by sloughing: usually in eight or ten days the membrane disappears and the ulcer heals. The fever is but slight, and the prognosis is good.

The organism found in these cases is the bacillus fusiformis, which is longer and broader than Loeffler's bacillus, stains best with methylene blue, and not at all with Gram's solution. It is pointed at the ends, bulging in the centre, and measures from 6μ to 12μ in length. It is generally associated with spirilla (*spirochæta denticolata*) or staphylococci. Local antiseptics, silver nitrate, and oxygenated water have been used in the treatment of the angina.

CHRONIC ENLARGEMENT OF THE TONSILS

Ætiology.—This is of common occurrence in children, without any apparent cause; some, it is true, are weakly in other ways, others maintain good health. Sometimes it can be traced to previous attacks of sore throat; on the other hand, those who have chronic enlargement of the tonsils are liable to temporary acute attacks. It often subsides as the patient approaches middle age, if not earlier.

Symptoms.—The tonsils are large, pale pink, lobulated on the surface, and firm in consistence. When only of moderate size, they may cause no symptoms. In other cases, the tonsils obstruct the passage of air from the nose through the pharynx, and the breathing

is at all times somewhat snoring. The child breathes with the mouth open, and the nasal passages being little used, the anterior nares are small, and the alæ compressed. A more remote effect is the production of pigeon-breast, from deficient expansion of the anterior part and bases of the lungs when the ribs are yet soft and yielding. Swallowing is laborious and clumsy, and speech is suggestive of something being in the mouth. Hearing is also deficient, from catarrh of the Eustachian tube; and taste and smell are said to be affected. The same symptoms may, however, be due to *adenoid* growths in the naso-pharynx (hypertrophy of the gland tissue in the upper part of the pharynx—the pharyngeal or Luschka's tonsil), which frequently co-exist with enlarged tonsils. Cough, nasal catarrh, restlessness, and headache are other symptoms observed in such cases.

Pathology.—The change in the tonsil is one of simple hypertrophy of all the component tissues.

Treatment.—The general health should be maintained by cod-liver oil, iron, and other tonics, including sea air. Local applications are of little service; but if the discomfort is considerable, the tonsils may be removed by the bistoury or tonsil-guillotine, after the local application of solution of cocaine. Adenoid vegetations are also removed by suitable forceps, or by a curette.

CHRONIC PHARYNGITIS

Ætiology.—Chronic inflammation of the pharynx may arise from repeated acute attacks, but more frequently results from certain injurious influences, such as the abuse of alcohol, excessive smoking of tobacco, and the continual use of the voice. It is constantly associated with a similar change in the soft palate, tonsils, or posterior part of the nose.

Symptoms.—The mucous membrane may be reddened, with dilated veins; in some cases there are numerous small gray elevations scattered over the pharynx (*granular pharyngitis*); in others small abrasions or ulcerations occur. The gray projections in granular pharyngitis are the enlarged follicles or mucous glands. In some cases the mucous membrane is covered with increased secretion, and the patient is constantly hawking and spitting; in others the surface is dry, and a certain amount of discomfort and difficulty in swallowing, with pricking pain and desire to cough, is the result.

Granular pharyngitis is often spoken of as a distinct affection. It may spread beyond the fauces proper to the top of the pharynx and to the larynx; the mucous membrane is in most cases dry, but sometimes the follicles are covered with viscid mucus. It may cause little or no discomfort; but there may be stiffness and dryness of the throat, constant desire to hawk and spit, and distress and difficulty in swallowing. The effort to talk is also

painful, and the patient may be obliged to stop to clear the throat. This condition of things is not uncommon in clergymen, public speakers, and others of like vocation, and has consequently been called "clergymen's sore throat." The symptoms are aggravated by exposure to cold, and an inherited disposition has been observed by some writers.

Treatment.—Local treatment is necessary in granular pharyngitis. Gargles are of little use, as they do not reach beyond the soft palate; but inhalations of alum or tannin may be employed, or the throat may be painted with astringent solutions, such as nitrate of silver (40 grains to the ounce), solution of perchloride of iron (℥j to ℥j), or tannin (℥j to ℥j), or iodised glycerine (iodine, 6 gr., pot. iod., 20 gr., ol. menth. pip., 5 min., glyc. to an ounce). If these fail, the granulations must be destroyed; and this is best done by the galvanic cautery or Paquelin's thermocautery, each nodule being successively touched. This may, of course, require several sittings; the resulting inflammation is checked by sucking ice for some hours afterwards. Anæmia, dyspepsia, and gouty tendencies must be met by suitable treatment.

RETROPHARYNGEAL ABSCESS

This, though chiefly a surgical complaint, requires short notice here, since it is apt to complicate the diagnosis of some throat complaints, especially laryngeal obstruction. It arises from caries of the spine, or more often from inflammation of the post-pharyngeal glands; and it forms a swelling in the back of the pharynx, which may press upon the larynx so as to cause dyspnœa and asphyxia. Thus it may be mistaken for croup or laryngeal diphtheria, but the cough and voice are not husky and hoarse as in the latter, but rather "gurgling." In a suspected case the finger should be passed to the back of the throat, when a fluctuated swelling will be felt. It should be opened by the surgeon.

DISEASES OF THE SALIVARY GLANDS

The salivary glands are liable to inflammation (parotitis) and to various new growths. Those which interest the physician are the enlargements of the organs by infiltration with lymphocytes, which are seen in cases of Hodgkin's disease, and lymphocytic leuchæmia.

Ptyalism, or excessive secretion of the salivary glands, results from mercurial poisoning, and occurs in some nervous conditions, as trigeminal neuralgia.

Xerostomia, aptyalism, or *dry mouth* is due to a deficiency of the secretion. In a mild form it is seen in diabetes, and various febrile

conditions; and it occurs rarely as an idiopathic condition which is very troublesome, and is only temporarily relieved by the use of pilocarpine. It has also been seen as a result of atrophy of the parotid glands.

PAROTITIS

Primary or *specific parotitis*, or mumps, has been already described among the infectious diseases (*see* p. 80).

Secondary parotitis is an acute form of inflammation which arises in the course of severe illnesses, such as pyæmia, fevers, dysentery, phthisis, and carcinoma. It occurs sometimes as a result of prolonged feeding *per rectum*. It may be metastatic in the case of pyæmia, but in most instances it is the result of infection from micro-organisms contained in the mouth, and reaching the gland through Stenson's duct. Suppuration is much more common in this form; several small abscesses are formed, and afterwards run together. They may discharge externally, behind the ramus of the jaw, or burst into the external auditory meatus, or burrow deeply down the neck, or behind the pharynx. Occasionally sloughing takes place.

The **Treatment** is that of acute local inflammations; fomentations will relieve pain, and when pus is recognised, an incision must be made; but recovery depends much upon the primary illness.

DISEASES OF THE ŒSOPHAGUS

ŒSOPHAGITIS

The œsophagus is much less liable than other parts of the alimentary canal to the various forms of inflammation. It may be injured by chemical substances or hot fluids, or inflammation may extend to it from neighbouring parts. *Chronic* inflammation results from the pressure of tumours, and from valvular disease of the heart. It produces thickness and opacity of the epithelium, or actual warty growths, or in some cases dilatation of the veins and desquamation of the epithelium.

OBSTRUCTION OF THE ŒSOPHAGUS

This is the most important pathological condition of this part of the alimentary tube. The causes are, impaction of foreign bodies, such as false teeth; compression from outside by mediastinal growths and thoracic aneurysms; the growth of cancerous or other tumours in the walls of the tube itself; constriction by the contraction of

ulcers following injury by corrosive poisons; and functional spasm of the muscular walls. The last three conditions will be separately considered.

CANCER OF THE ŒSOPHAGUS

This generally occurs in advanced life, and in males more often than in females. The growth occupies the middle and lower thirds of the œsophagus much more often than the upper third; but it is especially frequent opposite the bifurcation of the trachea, and is rare at the cardiac extremity of the œsophagus. It is always primary, usually of the epithelial variety, and of different degrees of consistence. In course of time it forms an irregular ulcerated surface on the inside. The tumour partially or completely encircles the tube, extending vertically from one to four inches; moreover, it often involves the trachea, or the root of the lung, or compresses the recurrent laryngeal nerves. The mediastinal lymph glands are enlarged, and not infrequently quite early the cervical glands.

Symptoms.—The first and prominent symptom is dysphagia. The patient finds he has difficulty in swallowing solids, when he may get fluids down with comfort. The difficulty increases gradually, and at length solid food has to be given up; liquids can alone be taken, and if more than a mouthful is attempted at a time it is regurgitated and the patient may choke. Pain is usually absent. After a few weeks the patient begins to emaciate, and loses strength and energy. The symptoms are generally progressive, but occasionally temporary improvement takes place from crumbling away of portions of growth from the surface, so as to enlarge again the calibre of the œsophagus. If no relief be afforded death takes place from simple exhaustion, or from complications. Thus, in some cases a communication with the trachea is produced by the spread of the growth; food-particles are inhaled, and a septic broncho-pneumonia is set up. In others, the lung is directly invaded by the new growth, and gangrene or broncho-pneumonia, with which pleurisy or empyema may be associated, carries off the patient. In others, again, compression of the recurrent laryngeal nerves leads to paralysis of the abductors of the glottis, which may produce asphyxia. Rarely a growth has eaten into the aorta and caused fatal hæmorrhage. Lastly, there may be deposits in other organs, especially in the liver and lungs. Occasionally, these are the cause of death, when the growth in the œsophagus has been too slight to produce any difficulty in swallowing.

Diagnosis.—Gradually increasing dysphagia in a person over fifty years of age is, in the great majority of cases, due to cancer of the œsophagus. Sometimes the fact of dysphagia may be overlooked: food may be retained sufficiently long in the œsophagus above the stricture for its regurgitation to be mistaken for vomiting, both by the patient and by a careless inquirer; and so a gastric lesion may be diagnosed. If the patient complains of vomiting immediately after food, he should be asked to drink in the presence

of the doctor, when it may be observed that he only takes a mouthful at a time, and waits as if expecting some to come back.

The presence of an obstruction is confirmed by the use of the *œsophageal bougie*, which will also show its position, and the extent of narrowing. The beginning of the œsophagus is six inches, and its junction with the stomach is sixteen inches, from the teeth. The bougie, however, does not discriminate between cancer in the walls and a tumour or aneurysm; the possibility of rupturing an aneurysm must always be borne in mind, and a careful examination for the symptoms of that disease should be made, and X-rays applied, before the bougie is used.

Direct inspection by the *œsophagoscope* is possible in some instances.

Auscultation of the œsophagus may also give assistance. It is effected by listening successively over each of the dorsal spines, while the patient swallows a mouthful of water, previously taken into his mouth. A peculiar gurgling sound is heard down to the point of the obstruction, but not below.

The presence of enlarged and hard cervical glands in a person suffering from dysphagia is in favour of malignant disease of the œsophagus.

Prognosis.—This is absolutely bad. Even if the obstruction is overcome, the malignant growth must be fatal by its further extension within a short time. Evidence of pneumonia at one base or marked fœtor of the breath show that the end is not far off. The duration is generally from six to twelve months.

Treatment.—If a bougie, even of small size, can be passed, the passage may be kept open for a time by its use every two or three days. But the maintenance of a canal for the food is best secured by some modification of the method of intubation introduced by Krishaber. A tube is passed through the stricture and retained *in situ* for several days or permanently: and the patient is supplied by that means with fluid nourishment. If these measures are inapplicable, the patient may be fed *per rectum*, or the stomach may be opened by the operation of *gastrostomy*. A diminution of the constriction is sometimes obtained by the use of radium applied locally to the growth in the œsophagus.

CICATRICIAL STRICTURE

In this again dysphagia is the main symptom; but it differs from cancer in this, that it may not advance beyond a certain point, and and that it does not lead to any secondary effects, except dilatation of the tube above it. In consequence of this dilatation food often accumulates above the stricture, and is regurgitated after a time.

The **Diagnosis** is generally determined by the history, and the absence of other symptoms. Cancer would be excluded if the patient were young.

Treatment offers a fair chance of success if the sound can be

passed through the stricture into the stomach. It should be used regularly once or twice daily, and attempts should be made to pass larger and larger instruments. Liquid food may be required always. In unfavourable cases gastrostomy may be advisable.

SPASMODIC STRICTURE

This occurs in nervous and hysterical young females, or even in males. There is difficulty in swallowing, accompanied by a painful sense of constriction in the throat and chest. The bougie may not pass at once, but steady pressure soon overcomes the difficulty. The neurotic condition of the patient must be treated.

DILATATION

Dilatation of the œsophagus will follow any long-standing stenosis; the enlargement is spindle-shaped (or *diffuse*), but often largest at the lower end. A few cases have been recorded of diffuse dilatation without any obstruction. Dysphagia and regurgitation are the main symptoms in both groups of cases; in the latter it is probably due to muscular paralysis, and feeding by a stomach tube is the obvious treatment.

DIVERTICULA

These are pouches in the walls of the œsophagus; they have been divided into (1) pressure diverticula, and (2) traction diverticula.

(1) *Pressure diverticula* arise from the impaction of foreign bodies, or from other local injury. As a consequenee, apparently, the muscular coat is weakened, and the mucous and submucous coats are bulged out between the muscular fibres, which do not share in the coverings of the diverticulum. When once this has taken place food accumulates in the sac, which gradually enlarges, so that it may attain a diameter of three or four inches. These diverticula are usually hemispherical in shape; they are most common at the back of the œsophagus in its upper part, and may project on both sides of the neck, sometimes on the left side only.

The **Symptoms** are dysphagia, regurgitation of food, and foul breath from the decomposition of food in the sac. So much food may accumulate as completely to obstruct the œsophagus. The presence of such a sac is sometimes detected by the bougie being at one time obstructed, at another passing easily into the stomach.

Treatment.—The pouch has been removed by operation. Apart from this, the patient should be fed through an œsophageal tube.

(2) *Traction diverticula* are caused by adhesion of the œsophagus to surrounding parts, whereby the coats are pulled out in a funnel-shaped manner. They have occurred in children as a result of suppuration of the bronchial glands. They may give an opportunity for the impaction of foreign bodies, but otherwise have no clinical symptoms.

DISEASES OF THE STOMACH

EXAMINATION OF THE STOMACH

In the healthy individual the stomach lies almost entirely within the bony thorax, only the pyloric extremity being exposed in the abdomen, and that is partly overlaid by the left lobe of the liver. The pylorus itself lies three-quarters of an inch to the right of the middle line. The organ is recognised by the full tympanitic note which is yielded on percussion of the epigastric region, and the lower part of the left thorax in front. This area is limited above by the præcordial dulness, posteriorly by the splenic dulness and pulmonary resonance, and below by the resonance of the colon and small intestines; but the loudness and extent of resonance vary with the distension of the stomach, and sometimes an obvious gastric note can be elicited over the base of the left chest behind. In health, the greater curvature when the stomach is full should not come lower than two fingers' breadths (one inch and a half) above the level of the umbilicus.

In disease, the resonant area of the stomach may be abnormally extended by dilatation, or it may be limited by the growth of a tumour near the pylorus, or by the enlargement of either liver or spleen so as to cover a greater portion of its area; it may be displaced downwards by tumours or liquid in the chest, and upwards by ascites, or by shrinking of the left lung.

It is, however, doubtful whether the limits of resonance can be safely taken to indicate the outline of the stomach. If it is empty the walls of the lower part are in contact. In a more distended condition, the resonance may still be simulated by that of the colon. Reliance has sometimes been placed on the method of auscultatory percusssion (*see* pp. 455, 673). A stethoscope is placed over the organ at different points, and the surrounding skin is percussed, and the limits of audibility of the resonant note mapped out: sometimes the skin is rubbed at different distances from the stethoscope, and areas of loud transmission of the friction sound are taken to correspond to the underlying stomach. Both these methods are untrustworthy. Distension of the stomach with gas by the successive introduction of 1½ drachms of bicarbonate of sodium, dissolved in 3 or 4 ounces of water, and 1½ drachms of tartaric acid in similar solution, will give useful information; but its distensive force will, of course, modify the shape and size of the stomach. Or the stomach may be inflated with air through a stomach-pump tube; or distended with a quart of water, so that differences in the areas of dulness and resonance may be observed; or a long sound may be introduced through the mouth, and its point pushed against the anterior wall of the

abdomen: in health a sound should not go in more than twenty-four inches.

The Röntgen rays have been successfully employed. The patient swallows a meal of porridge or bread and milk containing two ounces of a solid salt of bismuth, preferably the oxychloride. The rays are then used, and the position and size of the stomach are indicated by the shadow cast on the screen by the contained bismuth salt. An electric light can be introduced into the stomach, and the limits of the organ to some extent demonstrated (*gastro-diaphany* or *transillumination*).

The Gastroscope.—In special cases a knowledge of the interior of the stomach may be obtained by the use of this instrument. Like other forms of endoscope (bronchoscope, cystoscope), it consists of a metal tube, which can be passed into the cavity to be inspected: and the interior of this cavity is illuminated by an electric lamp and reflected to the eye of the observer by mirrors or prisms.

EXAMINATION OF THE CONTENTS OF THE STOMACH

Very important information may be obtained by the examination of vomited matters, and of fluids artificially withdrawn from the stomach during the process of digestion. We may thus try to ascertain the share which a deficiency of the acids, of the pepsin, or of the motor powers of the stomach may have in different forms of disease, especially the chronic disorders of digestion.

Vomited matters.—If the patient vomits, the quantity, odour, colour, and consistence of the fluid should be noted. The smell is usually acid, but it may be modified by substances recently taken, such as volatile oils or alcohol. The fluid may be colourless, or different shades of brown, or stained yellow or green by bile-pigment, or pink or red with blood. Often blood is altered by contact with the gastric juice, and a dark brown, opaque fluid is the result, resembling *coffee-grounds*. In consistence vomit may be watery, or thin mucous, or viscid or frothy. The presence of half-digested or undigested food should be noted, and the state of coagulation of recently ingested milk.

Microscopically, animal and vegetable tissues may be detected, such as muscle-fibres, cellulose, starch-granules, oil-drops, red blood corpuscles, leucocytes, and numerous micro-organisms, especially *torulæ, sarcinæ,* and sometimes Oppler-Boas bacilli. For chemical examination, the vomited fluid must be strained through fine muslin, and the filtrate may be submitted to the tests presently to be mentioned.

Test-meal.—If vomit is not available, the secretions and powers of the stomach are generally tested by the use of a test-meal. Either the stomach is first washed out (*see* Dilatation) or the test-meal is given in the morning on an empty stomach. Ewald's test-breakfast is $2\frac{1}{2}$ ounces of bread and 10 ounces of water or weak tea; and the

contents are withdrawn by the stomach-tube after an interval of one hour. The fluid is then analysed with reference to its total acidity by titration with an alkali, and to the respective amounts of hydrochloric acid, lactic acid, and of peptones it contains. Acidity due to lactic acid or to free hydrochloric acid can be detected by the use of papers steeped in a saturated solution of Congo red; the dye is turned blue.

One of the most trustworthy tests for *free hydrochloric acid* is that of Günzburg. The reagent consists of 2 grammes of phloroglucin, 1 gramme of vanillin, and 30 grammes of absolute alcohol. A few drops of this mixed with an equal quantity of a liquid containing free hydrochloric acid, and gently heated in a white porcelain dish, give a rose-red colour.

Uffelmann's test for *lactic acid* is as follows: Ten c.c. of a 4 per cent. solution of carbolic acid mixed with 20 c.c. of water, and a drop of tr. ferri perchloridi. The blue solution is turned yellow.

Butyric acid may be extracted by ether from the filtered stomach contents, and if the residue after evaporation of the ether is dissolved in water, the addition of a fragment of calcium chloride will cause the acid to separate in oily drops.

Acetic acid may be recognised by the claret colour it gives with tr. ferri perchloridi.

Hydrochloric and lactic acids can also be quantitatively estimated.

The *digestive power* or peptic properties of the removed fluid may be tested by its action upon measured portions of fibrin or white of egg, its acidity having been raised to the normal equivalent of ·2 per cent. by the addition of hydrochloric acid.

The *motor power of the stomach* can also be tested by the rapidity of disappearance of a test-meal. Thus 100 c.c. of soup, 60 grammes of beef-steak, and 50 grammes of white bread, should disappear in five hours (Riegel).

FUNCTIONAL DISORDERS OF THE STOMACH

Indigestion and dyspepsia are the terms which have long been used to indicate any such modification of the process of digestion as results in imperfect solution of food by the gastric secretions, delayed transmission of the chyme into the duodenum, or pain and discomfort in these processes. Such disorders are, of course, often caused by organic disease, whether inflammation or ulcer, or cancer, or mechanical obstruction; but they are much more often due to temporary disorders of secretion or motility, want of proper relation between the demands made upon the stomach's secretion and motility, and its power to respond, and finally to modified nervous conditions. The fault may lie in the improper quality or excessive quantity of food supplied, causing *gastric irritation*, or in deficient powers of the stomach, *atony* or *gastric insufficiency*. But the distinctions in individual cases are not always easy to establish; and,

moreover, gastric irritation of sufficient intensity or duration will result in inflammation, *gastritis*.

ACUTE INDIGESTION

This is a purely functional or mechanical disturbance, from the ingestion of food in too great quantity, or of specially irritating quality. Any one in perfect health may be tempted to take a larger quantity of food than his stomach can bear; or, with an ordinary quantity of food, some ingredient, such as ice or coffee, or alcoholic drink in excess, may be taken, which retards the process of digestion, and the whole quantity ingested remains for some hours in the stomach. Or the unexpected failure to digest may be due to preceding general exhaustion, in which the stomach shares; for instance, after excessive exercise in walking or climbing during several hours without refreshment, the stomach may fail entirely to digest even a moderate meal put into it.

Symptoms.—Either at once or within a few hours of the meal, there is a sense of distension and discomfort, or actual pain, in the gastric region; if the offending meal has been a late dinner the patient may go to sleep with little trouble, but wakes after a few hours with gastric distress, a dry tongue, and perhaps headache, and may lie wakeful for some hours. Sometimes a quick fluttering is felt from time to time in the præcordial region, or the heart actually intermits a few beats. In the morning there is little inclination for food, the tongue is dry and furred, and the skin clammy; but in the course of a few hours the symptoms subside. In other cases the illness is more quickly terminated by the occurrence of vomiting, and the stomach is generally emptied of the whole of its contents, which are only partially, if at all, digested, and are mixed with gastric mucus. The pain is often at once relieved. At other times the vomiting may be repeated, and bile, which has been regurgitated from the duodenum, may be discharged with the later efforts. Sometimes in the course of the next twelve hours the bowels are actively moved, from the passage into them of undigested or irritating materials.

Treatment.—Where the pain is severe, and the cause is obvious, immediate relief may be obtained from an emetic such as ipecacuanha or sulphate of zinc; if it fails to act, apomorphine by subcutaneous injection may be desirable. In milder cases it is sufficient to quench thirst with a very little ice, and to abstain from introducing anything further into the stomach until the distressing symptoms have subsided.

CHRONIC INDIGESTION

Ætiology.—The factors in chronic indigestion are the quantity and kind of the food, the secretory functions of the stomach, and the capacity of the stomach to circulate and in due time to expel its

contents. The last two factors are under the control, more or less directly, of the nervous system.

The process of digestion is impaired if the *food* be habitually excessive in quantity, or if it be improperly masticated from defective teeth, or from too great haste to swallow. Some foods are less soluble in the gastric juice, such as coarse-fibred meats, pulpy fruits, or stringy vegetables where cellulose is in excess. Pork, veal, game, and others mentioned under Gastritis are among these.

On the part of the *stomach* we may note that all the organic lesions are causes of dyspepsia; but that apart from these indigestion arises from excess or deficiency of gastric juice, from deficiency of hydrochloric acid, or of pepsin, or from excess of mucus. Weakness or degeneration of the walls of the stomach, and compression or displacement by adjacent viscera, the descent of the diaphragm, a pregnant uterus or other abdominal tumour may impair its motility, and thus prevent a proper admixture of the contents. The low position of the organ known as *gastroptosis*, which occurs especially in women, and which appears to be due in different cases to tight-lacing, a movable kidney, muscular strain, or relaxation of the abdominal muscles, as well as the above-mentioned pressure from above, may have a similar effect (*see* Glénard's Disease).

General illnesses and other causes of low vitality, such as anæmia, phthisis, and the infectious fevers, affect both secretion and motility.

Local Symptoms.—These vary much in different cases.

Local sensations.—Indigestion is shown frequently by pain in the epigastric region, which comes on after taking food, and lasts a certain time, gradually subsiding. It may be strictly localised, or radiate to the left, or extend to the præcordial region, when it is called *cardialgia* or *heartburn*, and is attributed to the contact of the acids of the stomach with the lower end of the œsophagus. Often it is felt between the shoulders, going "through to the back." Sometimes, as digestion slowly proceeds, the pain extends to the umbilicus or lower abdomen. In other cases pain begins when the stomach is empty, and is relieved by ingestion of food. Instead of pain there may be only a sense of discomfort, tightness, or fulness.

Gastralgia and *Gastrodynia* signify simply pain in the stomach, and are commonly used to designate gastric pain, from whatever cause arising. Such pains are often very severe, and spasmodic in character, but may be independent both of organic disease and of the digestive process, and are possibly neuralgic in origin. They occur often in females, where there may be good ground in the history and character of the patient for regarding them as neurotic: sometimes they occur in association with gout. An attack of gastralgia, again, may commence when the stomach is empty, and may be relieved by food.

Flatulence.—The excessive formation of flatus is a common result of dyspepsia. The gastric region is distended, much discomfort and

pain are caused, and waistbands may have to be loosened. Some relief is afforded by eructations, and after a time by the passage of the wind *per anum*. *Borborygmi*, or noises produced by wind passing along the intestine, occur at the same time. Flatulence and borborygmi also occur occasionally on an empty stomach. Flatus is sometimes due to bacterial fermentation of the contents of the stomach, as a result of a diminished secretion of hydrochloric acid, which normally prevents the action of bacteria; but it is also due to carbonic acid liberated from carbonates by hydrochloric acid. In other cases the air has been swallowed, or it enters the stomach from the intestines, or it is exhaled from the blood-vessels of the stomach.

Nausea is an occasional symptom of dyspepsia, and *vomiting* less frequent, except in drunkard's dyspepsia (*see* Chronic Gastritis). The vomited matter is either the ingested food, or merely mucus. With repeated emesis bile may be rejected, and a few streaks of blood; but a large amount of blood is quite rare. *Pyrosis*, or *water-brash*, is a name given to a condition in which a quantity of liquid is brought up into the mouth; there is burning pain in the epigastrium. The liquid is often neutral or alkaline in reaction, and is then commonly believed to consist chiefly of saliva; but it is sometimes acid.

General Symptoms.—The tongue is variable; it is often furred, and is large, pale, and flabby, or red, narrow, and pointed. The fur may be thin and white, or thick and yellow or brown. Constipation is frequent, but may be interrupted by occasional diarrhœa. Some cases are characterised by the rapid passage of undigested food through the intestines (*lienteria, lienteric diarrhœa*). The urine will vary with the amount of water ingested, and with the presence or absence of vomiting; and deposits, sometimes of urates, at others of phosphates, are likely to occur. The skin eruptions, urticaria, erythema, and acne, are often associated with indigestion, though the relations between them are not clear. The effect upon the body generally, or more correctly upon the nervous system, is seen in malaise, indisposition for exertion, headache, giddiness, subjective sensations of sight, drowsiness, irritability, and mental depression; while slight anæmia, or sallowness, some loss of nutrition, and in chronic cases a settled expression of discomfort or anxiety upon the face, are not uncommon. But in other instances there is no general indication whatever of the gastric fault.

Varieties.—In different cases it may be shown, as already implied, that the cause of a disordered digestion is either deficient or excessive motility, deficiency or excess of hydrochloric acid, or deficiency of pepsin; but these facts are often only ascertained after the examination of stomach contents or the use of test-meals. Some of the results of indigestion, such as the production of flatus and of acid eructations, give names to the trouble (*flatulent dyspepsia, acid dyspepsia*), but these results are not distinctive of their origin,

and a particular group of symptoms, *e.g.*, acidity or flatulence, may arise from many different remote causes, both functional and structural. It should be our endeavour to ascertain in each case whether the fault lies with the secretory functions, the motility of the stomach, or the nervous system.

The variety known as *atonic dyspepsia* (*gastric insufficiency*) arises in various conditions of depression of vital power, is common in anæmic persons, and appears to depend on a want of functional power of the stomach, in regard both to secretion and to muscular contraction. The tongue is broad, flabby, and indented by the teeth, there is little pain after food, except as a direct result of flatulent distension, and the appetite is bad, but there is no thirst. In some of these cases there is a deficiency of free hydrochloric acid. The slow digestion may be demonstrated by the test-meal.

Flatulent dyspepsia is a term which is given where the formation of flatus is excessive, and constitutes the prominent feature of the case; it is often a part of atonic dyspepsia, the result of insufficient motility of the stomach. It is largely aggravated by tea, coffee, pastry, green vegetables, and other foods not easily digestible. A common form of indigestion, in which flatulence is prominent, is one in which there is pain or wind after food; the pain is felt in the epigastrium, and goes through to the back; the tongue is thickly furred, and there is troublesome constipation; sickness is only occasional. In another form there are flatulence, distension, gastric pain, and constipation; but the pain does not go to the back, the tongue is clean, and there is never any vomiting.

In the condition called *hyperchlorhydria* there is uneasiness an hour or two after food, and this gradually increases to more or less burning pain, which may last two or three hours. The acid eructations which may accompany this are due to excess of hydrochloric acid, and not to the organic acids. The pains tend to be increased by starchy foods and diminished by foods richer in proteids.

Instead of being confined to the digestive period, the excessive formation of hydrochloric acid may be constant. This condition is called *gastro-succorrhœa* (Reichmann) or *gastroxynsis*. There is loss of appetite, with gnawing pain in the epigastrium, nausea and vomiting of large quantities of acid liquid. The attack lasts a few days.

By *Nervous dyspepsia* is generally meant a case in which severe gastric pain or vomiting or flatulence is the chief symptom in a patient of neurotic or hysterical tendencies, while digestion may sometimes be found by the test-meal to be perfectly performed (*see* Neuroses of the Stomach). But certainly the above perversions of secretion must depend sometimes on nervous defects, and hence come within the category of neuroses.

Treatment.—Indigestion requires for its treatment great care and judgment on the part of the physician, and perseverance and obedience to orders on the part of the patient. No pains are so readily forgotten as those of the stomach in the presence of appetite

or hunger. The first essential is that the cause should be considered, and this means that before regarding the case as one of functional indigestion, such organic diseases as ulcer, cancer, and dilatation should as far as possible be excluded. Causes *external* to the stomach should then be investigated. If teeth are defective or painful, the dentist's assistance must be sought. If the food is obviously unsuitable, either in quality or quantity, it should be modified. Moreover, the treatment will be more scientific if the exact condition of the secretions and of the motor power of the stomach can be ascertained by examination of vomited matter or of the contents withdrawn after a test-meal; and if the defects so ascertained can be adequately supplied. Where this is not possible or expedient, much may be done on principles that are still scientific. Indigestion, apart from organic lesions, is due either to irritating properties of the food, or to deficiency of secretion or motility of the stomach; and these indications may be met. The meals should be regulated; they should be taken at not too long intervals, and the food should be in moderate quantity. The more irritant and less digestible foods should be excluded, such as pork, veal, game, shell-fish, pastry, carrots, turnips, and parsnips. Boiled or roast mutton, fish, or chicken may be allowed; but in severer cases the diet should be confined to milk or peptonised milk, or milk and farinaceous articles. If acid fermentation is a marked feature, farinaceous foods should be limited, and milk and fish should be given. The liquid irritants, alcohol, tea and coffee should also be eschewed. Sometimes repeated experiment alone will show what foods the patient can tolerate: but constipation should be relieved; and the patient should avoid all mental worries and overstrain, or business anxieties. Where atony, debility, or nervous prostration is a prominent feature, complete rest is of great value; in other cases sufficient but not exhausting exercise should be taken. Where local atony appears to exist, but the patient is otherwise strong and vigorous, help may be gained by exercises developing the abdominal muscles, such as various forms of gymnastics, riding, and fencing; or abdominal massage and electricity. Various drugs are useful, especially the alkaline carbonates given before meals; bismuth; the mineral acids; the bitter tonics, calumba, gentian, and nux vomica; and carminatives, such as sp. ammon. arom., cardamoms, and ginger. Slight cases of oppression after meals are often benefited by dilute hydrochloric acid, which supplies the defective secretion in the stomach, and by nux vomica; in flatulence and constipation, rhubarb, soda and calumba, and rhubarb and magnesia are of value. Flatulence alone may be lessened by bismuth or sodium sulpho-carbolate before meals; by creosote, charcoal, or carbolic acid; or by ginger, peppermint, cardamoms, and other carminatives; and hyperchlorhydria by alkalies, especially sodium bicarbonate, given in 15 or 20 grain doses an hour or two after meals, or bismuth lozenges; while the food should be proteid rather than carbohydrate. As improvement takes place, nux

vomica or strychnia with quinine is useful in giving tone to the stomach and the system generally. In gastralgia or gastrodynia, and all cases with intense pain, opium or morphia may be administered in small doses, and belladonna liniment and hot fomentations should be applied to the stomach, or, in severe cases, a small blister.

NEUROSES OF THE STOMACH

As already implied, the neuroses of the stomach do not show themselves only in painful sensations, but also in modifications of the secretions of gastric fluids and the motor power of the stomach, on which perfect gastric digestion so much depends. *Gastralgia* and *gastrodynia* are sometimes no doubt independent of digestive defects, and rather neuralgic in their origin; but they are often difficult to discriminate from the pains of gastric ulcer, and genuine sufferers from the latter have often been wrongly regarded as hysterical. The *gastric crisis* of locomotor ataxy is another form of pain which, with its attendant vomiting, is a gastric neurosis. *Vomiting* is often hysterical or neurotic, and entirely independent of ulcer, cancer, or gastritis. It may occur directly food enters the stomach; it may be accompanied or preceded by pain. The known characteristics of the patient or the maintenance of health in spite of the symptoms may save one from errors. Children are not unfrequently the subjects of this difficulty. The vomiting of brain disease is another illustration. *Bulimia* or *hyperorexia* (excessive appetite) and *anorexia* (deficient appetite) probably have their seat in the stomach. Deficient appetite is common in hysterical females, and *anorexia nervosa* is an extreme form, already mentioned (*see* p. 416). *Flatulence* in some of its forms develops with such rapidity that it is not easy to explain it on chemical theories; and even its frequent relation to other neurotic conditions does not readily explain its mechanism.

Among neuroses of the stomach may also be considered the functional variations in secretion, and the excessive and defective motility already mentioned under Indigestion.

Treatment.—The more obviously the local symptoms are dependent upon the general condition of the patient, the more must the treatment be directed to the patient rather than to the stomach; and reference may be made to the sections on Hysteria and Neurasthenia for guidance. This applies especially to some forms of vomiting, of pain on reception of food, and of anorexia nervosa. Valerian or the bromides may be found useful in some of these conditions.

ACHYLIA GASTRICA

(*Apepsia*)

These names are given to a condition in which the gastric secretions, both pepsin and hydrochloric acid, are very deficient or absent: and this is due mostly to more or less atrophy of the gastric glands. It occurs in pernicious (Addison's) anæmia, as a result of gastritis, of local disease of the stomach, such as cancer, of general infections, and sometimes of nerve failure. The subjects of it are mostly over thirty years of age, and of either sex indifferently. Its existence is shown by the absence of hydrochloric acid from the gastric secretions, and their inertness when set to act upon egg-albumin (*see* p. 688).

Symptoms.—On the side of the stomach these are not distinctive; nausea, vomiting, eructations, gastric pain or distress, anorexia, or increased appetite occur in different cases: rapid emptying of the stomach often occurs. Headache, fatigue, muscular pains, and mental depression are the more common general symptoms.

Treatment.—This cannot be very hopeful if the glands are extensively atrophied, or if the cause is otherwise beyond cure. The introduction of pepsin generally does some good, and dilute hydrochloric acid should be given. As diet, koumiss, lightly boiled eggs, and small quantities of meat are recommended, but fatty foods should be avoided.

GASTRITIS

ACUTE GASTRITIS

Ætiology.—Acute inflammation of the stomach, or acute gastric catarrh, may be set up by various forms of irritants. The most intense form of gastritis occurs in poisoning by the strong mineral acids, or other corrosives (*toxic gastritis*). The more common cases arise in consequence of the use of indigestible food, such as lobster, crab, or shell-fish, or of unripe fruit, or of flesh, fish, fruit, vegetables, or other food which is in a state of commencing decomposition, and contains ptomaines or the bacillus enteriditis of Gaertner. It is thus frequent in hot weather, and may be further contributed to by the ingestion of large quantities of water. Infants frequently suffer in this way from unsuitable food, which is in different cases the mother's milk, or cow's milk insufficiently diluted, or one or more of the various starch-foods and biscuit-foods in use. Gastritis may also be due to various infections (*infective gastritis*): for instance, pneumococcal, typhoid, syphilitic, and tubercular forms of gastritis have been recognised.

Symptoms.—In corrosive poisoning the symptoms are briefly acute pain and tenderness in the epigastrium, vomiting of blood and

mucus, collapse, and frequently death. These cases are described in works on Toxicology.

In the more familiar cases of acute gastritis there is a feeling of weight, or oppression, at the epigastrium, and in many cases actual pain, which is increased by pressure, or by the ingestion of food. With this there are nausea and retching; or vomiting is at once produced by the introduction of anything into the stomach. The vomited matters at first consist of particles of food, afterwards they are watery or mucous, or stained with bile. There is no free hydrochloric acid: but later there may be organic acids due to bacterial fermentation. The patient is dull and heavy, with some headache, generally constipation, loss of appetite, thirst, an unpleasant taste in the mouth, a thickly furred tongue, and offensive breath. There is sometimes decided febrile reaction, and the pulse is soft and quick. Examination of the abdomen shows that the epigastric region is hard and tense, and sensitive to pressure. In many cases of food-poisoning the intestinal symptoms predominate (*see* p. 731).

In infants the complaint is generally associated with diarrhœa. The little patient is constantly fretting or whining from pain, the legs are drawn up to relieve it, the abdomen is tense and tender, food is either refused, or, if taken ravenously to quench thirst, is as rapidly rejected, emaciation soon occurs, and death may be the result. Except in the case of infants, the disease generally subsides in the course of a week or two, but repeated attacks may lead to a chronic condition.

Morbid Anatomy.—In the majority of cases nothing can be known of the condition of the mucous membrane of the stomach, since recovery takes place; and the changes which have been found in the stomach after death from the infectious diseases must not too readily be assumed as identical with those occurring in ordinary cases, since there is an absence of the symptoms characteristic of these last. But in the well-known case of Alexis St. Martin it was shown that changes quickly followed irritation of the mucous membrane. Red pimples appeared, which were sometimes filled with purulent matter, or there were red patches, or aphthous crusts, or abrasions. The gastric juice was secreted in less quantity, and mucus was poured out freely. Slight hæmorrhage also occurred sometimes. Ziegler states that in gastritis the mucous membrane is dark red and swollen, beset with small hæmorrhages, and covered with a film of mucus, mucoid epithelium, and extravasated leucocytes. The cylindrical epithelial cells of the gland ducts are in an extreme stage of mucoid change, and many desquamate; and the epithelial cells of the peptic glands are detached, and seem more granular than usual. The vessels of the interglandular tissue are distended; and the subglandular tissue and even the submucous layer are infiltrated. Abrasion and ulceration may also occur; often perhaps at the seats of previous hæmorrhages; and as later results, induration and atrophy of the mucous membrane supervene.

Diagnosis.—This is generally simple. It has, however, been pointed out that acute gastric catarrh may be confounded with the early stages of *enteric fever*, and with *peritonitis*. The former difficulty may not be cleared up until the appearance of rose spots and a typical diarrhœa, or reaction to Widal's test; in peritonitis we should expect more generally diffused pain, a more rapid onset, greater distension and early collapse.

Treatment.—It is of the first importance to give complete rest to the stomach. In severe cases food should be stopped altogether for a time, and as little as possible of any kind should be introduced into the stomach. If the symptoms do not abate in twenty-four hours, nutrient enemata may be employed. In milder cases very small quantities of milk and soda-water may be allowed, or peptonised milk, or koumiss; and thirst may be quenched by iced soda-water or seltzer-water, or small pieces of ice. For the pain, hot fomentations or poultices may be used, or, in very severe cases, leeches may be applied to the epigastrium, or opium in small doses may be given internally. The same drug will sometimes allay continued vomiting; bismuth and effervescing citrate of ammonium or potassium, or two or three minims of tincture of iodine in a teaspoonful of water given every half-hour, are also useful. Constipation may be relieved by enemas, or by a seidlitz powder, or other effervescing saline, if the nature of the case is obvious; but, in case of doubt, the former should be employed. Cases of gastric catarrh have been often treated with emetics, but these can only be advised when it is certain that the stomach contains a mass of undigested food, which is acting as an irritant. Washing out the stomach by syphonage may, however, often be useful at the beginning of an attack.

As the symptoms subside, the food may be gradually increased; milk in larger quantities and more often, then light puddings, dry toast, a little fish, and so on to the normal diet of health.

ACUTE SUPPURATIVE OR PHLEGMONOUS GASTRITIS

A small number of cases are on record of suppuration of the walls of the stomach, either in the form of a circumscribed abscess, or as a purulent infiltration. The abscess may be of the size of a walnut, a hen's egg, or larger; and is more often in the submucous than in the subserous layer; it may burst into the stomach or into the peritoneum. The cause may be pyæmic or puerperal infection, but has often been unexplained.

The **Symptoms** have been generally loss of appetite; severe pain in the abdomen, worst in the gastric region, and increased on pressure; vomiting, thirst, intense fever, and small irregular pulse; delirium, coma, and death. The vomiting has been generally bilious or mucous; in one case, pus was vomited from the ruptured abscess. Diarrhœa was often present. The symptoms resembled

in some cases peritonitis, in others pyæmia. The abscess has been sometimes felt as a tumour, when the diagnosis, otherwise difficult, becomes possible.

The **Prognosis** is bad, and much worse in the diffused form. Some cases have recovered after rupture of the abscess into the stomach.

The **Treatment** must be mainly symptomatic, unless the presence of a definite swelling should justify an operation.

CHRONIC GASTRITIS

Ætiology.—Chronic gastritis may be the result of an acute attack, but more often it arises from the continued ingestion of irritating or indigestible food, such as pork, veal, pastry, fruit, or tea and coffee in excess; and it is a constant result of undue indulgence in alcoholic liquors. Local conditions of the stomach may also cause it, such as the venous congestion which results from diseases of the liver and heart, and the irritation of malignant disease or chronic ulcer. Almost all conditions by which the processes of digestion and the preparation of the food for digestion are interfered with may be causes of gastritis, though they often do no more than induce the functional disturbance known as indigestion or dyspepsia. They are defective mastication, bolting the food, irregularity in taking meals, mental anxiety, overwork, and other debilitating influences, such as prolonged illness, fever, phthisis, or Bright's disease.

Morbid Anatomy.—In very chronic cases, the wall of the stomach is generally thickened, and presents various degrees of vascularity, not always very marked. Numerous dark or slate-coloured patches of pigmentation give evidence of former congestion or hæmorrhage; and occasionally small ulcers, *hæmorrhagic erosions*, are scattered over the surface. Sometimes the mucous membrane is atrophied entirely, at others there is a fibrous overgrowth of the interglandular, submucous, and intermuscular connective tissue, while the glands disappear or become cystic, and the muscular fibres perhaps waste. The process of thickening sometimes produces, especially in the pyloric region, a rough and wrinkled surface, commonly described as *mammillated*.

Symptoms.—There is generally some tenderness on pressure in the epigastric region, but pain is not often severe. It may be aggravated by food, and is felt in the epigastric region, and perhaps in the back between the shoulders, or there is a burning sensation internally. Nausea is more frequent, and there is sometimes vomiting. Vomiting is the most prominent feature of the gastritis of drunkards, and occurs in the morning immediately the patient rises from bed. The vomited matters mostly contain a good deal of mucus, but rarely blood; sometimes they are acid, and contain butyric, lactic, and acetic acids from fermentation in the stomach, but the hydrochloric acid is deficient. Flatulent distension of the

epigastric region, and eructation of gas may also be present. The associated conditions are decided thirst, capricious and often deficient appetite, offensive breath, and unpleasant taste; a furred tongue, red at the tip and edges, narrow and pointed, but sometimes broad and flabby; and red or spongy gums, and cracked lips. The bowels are, as a rule, constipated; but they may be loose or altogether irregular in their action. The urine is variable, often scanty, acid, high-coloured, and depositing urates; sometimes paler, feebly acid, and depositing phosphates on boiling. There is sometimes slight febrile reaction, or a feeling of malaise; sleep may be disturbed, and the patient is nervous or depressed. In prolonged cases there may be emaciation.

Diagnosis.—It has been usual to contrast chronic gastritis with *atonic dyspepsia* (*see* p. 692), in which the indications of inflammation are absent, and those of debility, depression, and anæmia predominate, whereas in gastritis the signs of irritation and mild inflammation are present. The important features are the slight fever, the condition of the tongue, the local tenderness, the vomiting of much mucus, the diminished amount of hydrochloric acid, and the absence of other evidence in favour of ulcer or malignant disease. The fluid withdrawn after a test-meal shows badly digested food, the presence of much mucus, diminished total acidity, diminished free acid, and often absence of free hydrochloric acid.

The possibility that a patient who says he vomits directly he takes food, is really regurgitating food from an obstructed œsophagus must be remembered (*see* p. 683).

Prognosis.—Chronic gastritis is often troublesome, but recovery may be expected with persistent treatment.

Treatment.—It is of the first importance to deal with the causes which have led to gastritis. Perfect hygienic conditions should be secured in the way of residence, exercise, occupation, and regularity of meals. The food should be bland, though nutritious; all the more indigestible kinds should be eschewed, and tea, coffee, and alcohol should be left off entirely.

In severe cases it is well to begin with the simplest possible diet, such as milk, or milk and farinaceous articles: after a time, as the symptoms subside, fish may be added, and then mutton or beef, chicken, mashed potatoes, cauliflower, and the less fibrous green vegetables. Pork, veal, game, shell-fish, pastry, carrots, turnips, and parsnips are foods which should be avoided. As the bowels are generally constipated, they should be kept open by an occasional dose of Friedrichshall or Hunyàdi János water, Carlsbad salts rhubarb and magnesia, aloes and sulphate of soda, or some of the other laxatives mentioned under Constipation. The medicinal remedies of most value are bismuth subnitrate, or the liquor bismuthi, sodium bicarbonate, and the vegetable bitters, gentian or calumba. The alkalies may be given before meals to stimulate acid secretion. Benefit may also be derived from the dilute mineral acids, hydrochloric and nitro-hydrochloric, supplying the deficiency

already noted; and they may be combined with nux vomica, or strychnia and the bitters. Certain symptoms may require special attention. For persistent vomiting one may give effervescent saline remedies, dilute hydrocyanic acid, oxalate of cerium, or tincture of iodine (3 to 5 minims in 2 drachms of water every hour). For flatulence, bismuth before meals is well suited, or aq. menth. pip., sp. armoraciæ co., creosote, carbolic acid or wood charcoal. Severe pain may require locally hot fomentations or a small blister, or opium or morphia internally.

DILATATION OF THE STOMACH

Dilatation of the stomach may take place very gradually (chronic dilatation), or may occur quite suddenly (acute dilatation). The former is much more frequent, and will be described first.

CHRONIC DILATATION

This results (1) from the various conditions which produce obstruction of the pylorus; and (2) from conditions which alter the contractile power of the muscular walls. The causes of obstruction are most often cancer of the pylorus; cicatrices of ulcers of the pylorus or duodenum less commonly; hypertrophic stenosis of the pylorus; pressure from without, binding down by adhesions, or dragging of a prolapsed kidney; and, quite exceptionally, cicatrices from corrosive substances, which, however, generally involve the œsophageal aperture.

The causes of weakening of the muscular tissue are chronic inflammation (gastritis), excessive overloading of the stomach, such as sometimes occurs in the insane, in drinkers and in gluttons, excessive formation of gas from whatever cause arising, and the interference with its nutrition which occurs in prolonged fevers and anæmia. Obstruction produces the greatest extent of dilatation.

Physical Signs of Dilatation.—In marked cases, when the abdomen is exposed, it is seen to be asymmetrical, presenting a rounded *prominence* in its left half. This prominence extends below the level of the umbilicus, its lower margin having a curve convex downwards and outwards, from the lower part of the costal margin to the right of the middle line. The left half of the epigastrium may be sunken above another shorter curved line, which corresponds to the lesser curvature of the dilated stomach; but the visibility of the lesser curvature is regarded as evidence of downward displacement of the stomach (*proptosis*), which often accompanies dilatation. From time to time a wave of *peristaltic movement* passes from left to right and downwards across the prominent part. A portion at the extreme left, about the size of the palm of the hand, quickly forms a convex prominence, with a decided amount of resistance to pressure; in a few seconds the swelling subsides, and

another part, more to the right, swells up for a similar length of time. After each successive portion of the stomach-wall has become hard and prominent the whole subsides. This phenomenon occurs spontaneously, or may be set up by manipulating the abdominal wall, or flicking it with the finger sharply, or sometimes on mere exposure of the abdomen. The enlargement of the stomach both downwards and towards the right side, may be demonstrated by one or more of the methods described above (*see* p. 686).

Percussion of the swelling gives varying results, according to the proportions of air and liquid which the dilated stomach contains; generally the lower part is dull, the upper part is tympanitic; and some change in the relative positions of the two sounds may be obtained by altering the position of the patient, the dull area always being the lower. By sharp movements of the abdomen, as when the patient is shaken, or the prominent stomach is roughly manipulated (best by suddenly pressing upon it, and quickly withdrawing the hand), the liquid contents are set in motion, and *splashing* can be heard and felt, or heard on application of the stethoscope to the left hypochondrium. This, however, has no significance unless it can be recognised over an abnormal area, as, for instance, as low as an inch from the umbilicus, or at a time when normally the stomach should be empty, namely, six or seven hours after a meal.

A striking feature of many cases of chronic dilatation is the manner in which vomiting takes place. The food is retained for three or four days, and then two or three pints of fluid are vomited at once. It is generally of a grayish-brown colour, frothing on the surface; and on microscopic examination it shows numerous spores of the yeast-plant, *torula cerevisiæ;* the spores, in groups of four, known as *sarcina ventriculi,* so called from their resemblance to boxes or bales tied round with a cord; and long rod-shaped bacilli, the *Oppler-Boas bacilli.* In other cases the vomiting is more frequent, and the quantity ejected is less at a time.

In addition to the vomiting, the patient suffers from discomfort or actual pain, which is increased as the contents accumulate, and is temporarily relieved after they are evacuated. Great thirst, loss of strength, emaciation, pallor, and constipation are also observed. Much mental depression, and sometimes tetany and convulsions, may also occur. The urine is scanty, and may contain acetone or acetic acid.

Diagnosis.—This depends upon the physical characters above detailed, of which visible peristalsis is the most conclusive. A simple proptosis may be distinguished from dilatation if the lesser curvature be recognised and compared with the lower limits of the organ. The cause of the dilatation must be determined by the history and by the presence or absence of pyloric thickening or tumour.

Prognosis.—Dilatation from narrowing of the pylorus must persist as long as the disease which causes obstruction; and treat-

ment other than surgical can only be palliative. When the distension results from weakening of the muscular walls, the outlook is more favourable, and recovery may take place.

Treatment.—For dilatation of the stomach, the operation of washing it out (*lavage*) has been found of great value. The overdistended organ is thus relieved of the accumulation of liquid and undigested food; and any catarrh which may co-exist is at the same time benefited. A rubber tube attached to a funnel is introduced into the stomach; the stomach is filled by raising the funnel above the level of the mouth and pouring in water; it is emptied again by depressing the funnel, and inverting it into a suitable vessel. Or the tube in the mouth may be connected by a Y-shaped joint with one tube passing upwards to a funnel, or other receiver, and another downwards into a vessel; when water is poured in, the lower tube is closed by the fingers just below the joint, and when it is desired to empty the stomach the upper tube is compressed and the lower left free. The stomach is first emptied entirely of its contents, and is then rinsed out, one or two pints of water being introduced and removed; and the process is repeated till the contents come out nearly clear. The water used for washing it is either pure, or contains bicarbonate of sodium (1 or 2 per cent.), or salicylic acid (1 per cent.). The washing should be done once daily, half an hour before the largest meal.

Food should be given in small quantities and at short intervals. Starchy and saccharine foods should be restricted or even prohibited, to prevent fermentatation; and liquids should not be given during digestion, or even at all. To compensate for this, they may be injected into the rectum. Tender meats, meat essences, minced meats, and a little fat or cream may be given. The bowels should be kept active, if necessary, by salines, such as magnesium sulphate and Carlsbad salts.

If the obstruction is organic (cicatrised ulcer or cancer) an attempt should be made by surgical means to widen it, or remove it; or a gastro-enterostomy should be performed.

ACUTE DILATATION

Cases of this kind are rare: two were described many years ago by Hilton Fagge, who attributed them to an acute paralytic distension. Campbell Thomson has collected 44 cases from various sources. Their occurrence is not easily explained; they are generally of very sudden onset. In the majority of cases there is no obvious cause of obstruction, but some have come on after overloading the stomach, a few after injury, and more than one-fourth after surgical operations.

The patient is seized with vomiting, and brings up frequently large quantities of green, brown, or gray fluid. With this are gastric discomfort, pain, and tenderness. The abdomen is generally found to be considerably swollen in its left and lower portions,

while the epigastrium is relatively flat. Visible peristalsis is quite exceptional (once in 44 cases); but varying amounts of resonance, fluctuation, and splashing may be obtained. The patient becomes collapsed, suffers from thirst, the urine is scanty, and the bowels are confined. Though the vomiting may cease for a time, and apparent improvement ensue, the prognosis is very bad, and death may take place in spite of mechanical or surgical assistance. The symptoms may last a few days.

After death the stomach is found to be enormously distended, stretching down towards the pubes, and there bent on itself with a portion returning up towards the duodenum. The distension sometimes extends some way along the duodenum.

The pathology is obscure; but the facts seem to be in favour of a primary paralytic distension followed by profuse secretion.

Treatment.—The stomach should be washed out at once; and the evacuation may be aided by turning the patient on his face. Food should be administered by the rectum. Failing these measures, Thomson suggests a gastro-jejunostomy.

HOUR-GLASS CONTRACTION OF THE STOMACH

This condition, at one time thought to be congenital, is now regarded as always due to acquired lesions, viz., either perigastric adhesions, or cicatrising ulcer, or cancer (Mayo Robson, Moynihan). The constriction is generally 3 or 4 inches from the stomach, which is thus divided into a cardiac or proximal, and a pyloric or distal cavity. In this situation the constriction forms an obstruction, causing dilatation of the cardiac pouch, with the symptoms attaching to this. By the use of lavage and carbonic acid gas generated in the stomach, or bismuth and the Röntgen rays (*see* p. 687), the presence of a second (pyloric) pouch can be sometimes shown. Thus, in lavage, more fluid may go in than comes out, the surplus being lost in the distal half: or after thorough washing out, fresh fluid appears, with an offensive odour; or there is splashing in the distal pouch after the cardiac half has been thoroughly washed out; or with carbonic acid gas two areas of distension are seen with a line of constriction between them. But these tests may all fail.

The most efficient treatment is surgical; the constriction can be widened, or the proximal pouch can be united with the distal pouch, or with the jejunum.

ULCER OF THE STOMACH

Ulceration of the stomach occurs in several forms. The slighter forms of ulceration occur in the course of chronic gastritis, and in consequence of hæmorrhage into the mucous membrane. These last are described as *hæmorrhagic erosions*, and they are the results

of congestion in cardiac disease, in emphysema of the lungs, in portal obstruction and in infectious disorders. But the form which has the greatest clinical importance, often known as the round or perforating ulcer, is generally thought to be primary, and occurs in two forms, *acute* and *chronic*.

Ætiology.—Ulcer of the stomach appears clinically to be much more frequent in women than in men, in the proportion of at least three to one, but it is fatal to the sexes in about equal proportions. The explanation is that acute ulcer is very common in young women between fifteen and thirty, and frequently heals; whereas in men chronic ulcer is more frequent between thirty and fifty or sixty. Another explanation may be that cases of hæmorrhage by oozing, without ulceration, in young women have been wrongly taken for ulcer. Either form is rare in children. Ulcer is seen more often in the poorer classes, and largely among female servants; it is also associated often with chlorosis and anæmia, but beyond this its predisposing conditions are not very obvious. Menstrual disorders have also been credited with an influence; but it is suggested that the abdominal congestion of menstruation has more to do with it than the fact of amenorrhœa.

Morbid Anatomy.—The acute ulcer is from half to three-quarters of an inch in diameter, with sharply defined, clear-cut edges, and soft walls, of more or less funnel shape, and closed at its base by the submucous, or muscular or peritoneal coat according to its depth. The chronic ulcer is generally much larger, and may reach a diameter of five or six inches. It extends deeply into the wall of the stomach; the edges are thickened and raised, from infiltration with inflammatory fibroid material, and overhang the ulcerated surface; and the thickening extends some little way into the surrounding mucous membrane.

Gastric ulcers are often solitary; this is especially the case with the chronic ulcer, which is single in about four-fifths of the cases; whereas the acute ulcer is multiple in more than half the cases.

The position of the ulcer is of importance; in more than half the cases it is in the neighbourhood of the pylorus, but this is mainly on account of the preference of the chronic ulcer for this site; the acute form is found with almost equal frequency in the pyloric region, the middle, and the cardiac region. Ulcers are also much more frequent on the posterior than on the anterior surface; and near the lesser than near the greater curvature.

When the ulceration reaches the peritoneum this may rupture so that perforation takes place, the contents of the stomach escaping into the peritoneal cavity and setting up intense general peritonitis, or a more localised abscess, *perigastric abscess* or *subphrenic abscess*, or, if it contain gas, *subphrenic pneumothorax;* and this abscess may perforate the diaphragm and set up pneumonia, pleurisy, or pericarditis; or it may perforate the colon or duodenum, or open again into the general peritoneal cavity. More often the inflammatory process, extending to the serous surface, causes the stomach to

adhere to one of the adjacent parts before perforation can occur. This is most frequently the pancreas or the left lobe of the liver, but adhesion also takes place occasionally to the diaphragm, spleen, colon, anterior abdominal wall, and even the suprarenal capsule. The ulcerative process then extends into the newly attached organ, and large cavities may be formed in the liver and pancreas. Thus also the diaphragm may be perforated, with subsequent pleurisy and pneumonia; and the colon may be opened, or the abdominal wall invaded, with the formation of *gastro-colic fistula* in the one case, and *gastro-cutaneous fistula* in the other. Rarely, an old ulcer causes so much adhesion and matting together of the parts, that a cancerous tumour is closely simulated. Hæmorrhage is a common accident, mostly from gastric vessels in the wall of the ulcer; but sometimes from the splenic artery after adhesion to, and ulceration of, the pancreas.

But many ulcers recover completely, and small scars are often found. Larger scars, which are thick and puckered, may themselves give rise to considerable trouble. Thus, at and near the pylorus they may by their contraction cause *stenosis*, and consequent *dilatation of the stomach;* if near the cardiac extremity, the stomach may be contracted. Sometimes an *hour-glass contraction* is due to ulcer. Ulcer may set up a chronic gastritis, or rarely a phlegmonous gastritis; the adhesions to surrounding parts (*perigastric adhesions*) sometimes give rise to pain and dragging sensations; and lastly, an old ulcer may become the seat of cancerous growth.

Pathology.—The most generally accepted view with regard to the origin of gastric ulcer is that it is due primarily to a digestion by the gastric juice of some portion of the mucous membrane which has become anæmic, degenerated, and finally necrosed; and the constant over-acidity of the secretions is in favour of this. The causes of this local necrosis are probably numerous, such as embolism thrombosis, and degeneration of a small vessel; small hæmorrhages due sometimes to portal congestion, sometimes to hyperæmia during menstruation; inflammation of the solitary glands after fevers (Fenwick); bacterial necrosis (Martin); rarely injury, pressure, or chemical agents. The influence of acid secretions in the production of ulcer is illustrated by the occurrence of duodenal ulcers chiefly in the part which is proximal to the entrance of the pancreatic duct, and therefore exposed to the unneutralised acid chyme; and by the occurrence after the operation of gastro-jejunostomy of ulcers in the jejunum, a part of the intestine less liable than any other to ulceration. More recently explanations have been looked for in (1) the action of specific *gastro-toxins;* (2) destruction by embolus or hæmorrhage of a supposed *antipepsin* in the gastric cells, which normally protects them against peptic action. The recognised relation to anæmia may be that in the anæmic person both hæmorrhage takes place more readily, and the damaged tissue sooner yields to the action of the secretions.

Symptoms.—In a large number of acute cases the first symptom is *hæmatemesis,* or vomiting of blood which proceeds from the ulcer; in some others the symptoms are the pain and vomiting which occur in the chronic form; and in a very few *perforation* of the ulcer takes place into the peritoneum as the first sign. In hæmatemesis the blood may be extravasated in large quantity at once, flowing freely from a large artery, so that it is promptly vomited, unmixed with gastric contents, and retaining its arterial brightness. The patient, who may have never brought up blood before, feels faint, has a sense of oppression in the epigastrium, and in a few minutes vomits the blood, which may amount to one, two, or three pints.

Some of the blood discharged into the stomach finds its way into the intestine; the hæmoglobin is converted into hæmatin, and the motions subsequently passed are black, treacly, or tarry, constituting *melæna;* these may appear some hours after the hæmatemesis has ceased. The vomiting of pure blood may continue so as to be fatal; more often it ceases entirely, and may not be repeated. High degrees of anæmia and weakness result from the loss of blood. Occasionally, when hæmorrhage occurs no blood is vomited, but the whole passes *per rectum.* Fenwick points out the frequency with which hæmorrhage from a gastric ulcer occurs secondarily in septic conditions, such as pyæmia, pneumonia, typhoid fever, and erysipelas.

In *chronic* ulcer also the first symptom may be hæmatemesis, but in most cases the symptoms are pain and vomiting.

The *pain* is situated in the epigastrium, just below the ensiform cartilage, sometimes nearer the umbilicus, or to the right or left of the middle line—the right more often than the left. It is generally brought on by the ingestion of food, appearing from half an hour to two hours after a meal; it may continue intense until vomiting takes place, by which it is generally relieved, or it subsides as the food leaves the stomach. In character it is pressing, boring, tearing, or burning, and more severe than in any other gastric disorder. Sometimes there is pain in the back, between the eighth dorsal and the second lumbar vertebræ. There is often tenderness and hyperæsthesia over the epigastrium.

The *vomiting* is nearly always determined by the food; and the matter vomited consists of food, and contains an excess of hydrochloric acid. Occasionally blood is present in small quantities, and mixed with the contents of the stomach, so that the hæmoglobin is converted into hæmatin by the acid gastric juice; and the vomited matter has the turbid, blackish-brown appearance which has been compared to *coffee-grounds.*

The continued pain, the defective assimilation of food from vomiting, and the loss of blood, naturally impair the general condition of the patient sooner or later; but there is no fever, the tongue is clean, unless there is much gastric catarrh, and the appetite is often very good. Constipation, however, is frequent.

Examination of the abdomen generally reveals nothing; there may be some hardness or tenseness of the abdominal walls. Only in the case of old ulcers with much thickening, or adhesion to other organs, can anything like a tumour be felt; and if pyloric stenosis results, the dilated stomach may be recognised. The hydrochloric acid of the stomach contents is often in excess; but sometimes it is diminished. After hæmatemesis there is a characteristic pallor, with the usual hæmic murmurs over the præcordia (*see* Anæmia).

In some cases there are symptoms differing but little from those of gastric catarrh or other form of dyspepsia, and consisting of pain or discomfort after food, distension, flatulence, nausea, and occasional vomiting.

The symptoms of gastric ulcer are undoubtedly very amenable to treatment, and *post-mortem* results as well as clinical records show that recovery often takes place; this, however, generally requires the continuance of treatment, and especially the judicious avoidance of harmful ingesta, for a very long period. Many patients, after months of freedom from symptoms, are again severely affected. In the more serious cases pains and vomiting are constant, and much blood is lost in the coffee-ground matters ejected. The chief cause of death is perforation, leading to general peritonitis, or to a localised peritonitis which may terminate in the ways mentioned (*see* p. 704); the other causes are exhaustion from continuance of pain, and vomiting, especially when dilatation follows a pylorus constricted by a healed ulcer; large hæmorrhages; and intercurrent diseases like tubercle, pneumonia, or heart disease.

Diagnosis.—Hæmatemesis is an important element in the diagnosis. Pain, vomiting, flatulence, &c., may be quite inconclusive, until the discharge of a large quantity of blood gives evidence of the ulcerating process. Hæmatemesis occurs also in cirrhosis of the liver and in cancer of the stomach; *cirrhosis* may often be shown by the age of the patient, the known alcoholic habits, and the facial aspect. Drunkards also suffer from gastric disturbance, but mostly in the form of vomiting or retching in the morning, when the stomach is empty; nor is pain a prominent feature. The hæmorrhage of *cancer* is rather in small quantities often repeated, than in large quantities at one time. A tumour is generally present, and the cancer patient is rarely under forty years of age. Hæmorrhage may also occur from the gastric mucous membrane through the minutest possible *erosions* of the surface, scarcely deserving the name of ulcer; and in some cases the blood escapes as a general *oozing* from the surface without either ulcer or erosion. Such cases, called *gastrostaxis*, occur in young women, of whom some have no accompanying symptoms, and others have some gastric pain and tenderness; but there is some difference of opinion as to the frequency with which such bleeding takes place apart from minute ulcers. Of course,

hæmatemesis must not be confounded with hæmoptysis (*see* pp. 527, 536), nor with vomiting of blood after epistaxis.

If blood is not obvious in the vomit or fæces, it may yet be present in sufficient quantity to be detected by chemical tests; and the possibility of this *occult blood* may always be considered. It may be shown by the guaiacum test and by the benzidine test. Whether vomit or fæces, it should be treated with glacial acetic acid, and extracted with ether to destroy other substances which may also react; or a piece of solid fæces, the size of a pea, may be placed in a test-tube with 4 c.c. of water, and stirred with a glass rod; the tube is then lightly stopped with cotton-wool, and the contents brought to the boiling-point

Guaiacum test.—To the fluid in a test-tube a drop or two of tincture of guaiacum is added, and then 2 c.c. of ozonic ether, when a bright blue colour appears.

Benzidine test.—A few grains of benzidine are dissolved in 2 c.c. of glacial acetic acid, and ten drops of this are added to 3 c.c. (or a drachm) of solution of peroxide of hydrogen (*e.g.*, dioxygen). To this mixture two or three drops of the fæcal fluid are added; and a green colour is produced if blood is present.

That this may be regarded as a proof of occult hæmorrhage, the patient must have abstained from blood-containing foods, *e.g.*, meat, for forty-eight hours beforehand.

If there is no evidence of hæmorrhage, the diagnosis is not always easy; sometimes ulcer can only be suspected. A severe localised pain of weeks' duration, aggravated by food, relieved by abstinence or judicious diet, accompanied by local tenderness, a clean tongue, and absence of general disturbance other than can be explained by the pain and vomiting, in a patient between seventeen and thirty-five, are grounds for the diagnosis and treatment of gastric ulcer. The diagnosis from cancer will be further considered below (*see* p. 715).

Prognosis.—This must always be doubtful, since neither hæmorrhage nor perforation can be foreseen. The earlier a case comes under treatment the more likely it is to be favourable. A long duration of severe dyspeptic symptoms and frequent coffee-ground vomit are unfavourable. A profuse hæmorrhage following upon pronounced symptoms suggests deep ulceration, but its very severity may compel a thorough course of treatment, to which the patient otherwise would not have submitted. Dilatation of the stomach is, of course, unfavourable.

Treatment.—The most important indication is to give the stomach as much rest as possible. For at least three weeks the patient should be in bed, and for some weeks afterwards should take but little exercise. If the pain is severe and vomiting is frequent, food should be given for a few days entirely by the rectum. After a time, or from the beginning in the less severe cases, it may be given by the mouth. This should be at first nothing more than milk, an ounce or two every two or three hours; if this is not well

borne it may be mixed with one-third of its quantity of lime-water or of soda-water; or, better, it may be milk peptonised by the addition of the liquor pancreaticus of Benger. In any case, the quantity introduced at one time into the stomach must be small, and such as will not induce pain or vomiting. The food may afterwards be increased, so that the daily amount gradually rises from one pint to two or three pints in the twenty-four hours. In the third week the milk may be thickened with arrowroot, ground rice, or biscuit powder; and beef-tea or other meat solution may be added. After another week or two, if the symptoms have entirely subsided for several days, more solid food may be allowed, and this may begin with fish, chicken, or raw pounded meat. Vegetables, fruit, and pastry must be avoided almost throughout.

A method of treatment has been introduced by Lenhartz, whereby it is sought to introduce more nutriment, both proteid and fatty, at an early stage of the illness; and it is claimed that it can be done quite safely, even after a hæmorrhage, while it obviates the annoyance of rectal feeding. Complete rest is necessary, and an ice-bag is placed over the epigastrium. The diet consists of eggs beaten up with milk and sugar. On the first day one egg is given with six or eight ounces of milk, and each day one egg and four ounces of milk are added until the patient is taking six or eight eggs in twenty-four hours with two pints of milk. This amount is continued through the second week. At the end of the first week one ounce of raw minced meat is given, and a little more on successive days; on the seventh or eighth day boiled rice is added, and later softened bread, bread and butter, and pounded fish, which gradually in later weeks replace the eggs.

It has been shown that fats will depress the secretory activity of the stomach; and accordingly oil and cream have been used with some success in the treatment of gastric disorders, where acid secretions are in excess, including gastric and duodenal ulcers. Cream may be usefully added to the milk diet: and the diet of Lenhartz contains much fat in the yolk of eggs. Oil has also been given separately, as one ounce of olive oil, or of almond oil, or of an emulsion, before each meal, with some success; but it is not well received by all patients.

For pain, if unrelieved by diet, one may give opium in small doses of the extract or tincture, or the liquor morphinæ hydrochloratis in 10 or 15 minim doses. In severe cases the hypodermic injection may be used; but the opiate treatment must always be discontinued as soon as relief is obtained. Bismuth is also of value; 10 to 30 grains of the carbonate suspended in mucilage should be given before meals. Much larger quantities have been given recently with success, for instance 2 or 3 drachms in a single daily dose with plenty of water before breakfast. Local applications to the epigastrium may be used, such as hot fomentations, mustard leaf, or even blisters in severe cases; sometimes ice-compresses give relief. Heartburn may be relieved by alkalies, especially

bicarbonate of sodium; constipation by cold water enemata, or Carlsbad salts before breakfast, or by compound liquorice powder, or pills containing rhubarb or aloes. Vomiting may be checked by morphia or bismuth and morphia, by effervescing medicines, or by tincture of iodine (♏ij in water ℥ij every hour).

If a profuse hæmorrhage occurs the patient must be kept at rest, and no food must be given by the stomach for some hours; ice should be applied to the epigastrium. With continued discharge of blood, adrenalin may be given (20 or 30 drops of solution of 1 in 1000), or other astringents, such as tannin, acetate of lead, calcium chloride, alum, or solution of perchloride of iron in 5-minim doses every hour. When death has been threatened, life has been saved by the surgeon opening the stomach and suturing the bleeding vessel. Gastro-enterostomy is also useful under similar conditions. But operation is of no value when there is general oozing; the treatment must depend on rest and astringents or hæmostatics.

If a patient known to suffer from gastric ulcer is seized with the symptoms of perforation (*see* Peritonitis) the abdomen should be opened as soon as possible—*i.e.*, within a few hours—the peritoneal cavity washed out, and the ulcer sutured. In ulceration of old standing, with frequent recurrence of pain and vomiting, or of severe hæmorrhages, or with evidence of much thickening about the ulcer, an operation is justified. The ulcer may sometimes be excised; more often the best plan is to perform a gastro-jejunostomy in order to give rest to the ulcerated surface.

Stenosis of the pylorus causing dilatation of the stomach will also require operation. Gastro-enterostomy is more frequently performed, to provide a ready passage into the bowel; but sometimes the contracted pylorus can be stretched from within (Loreta's operation), or widened by the operation of *pyloroplasty*.

ULCER OF THE DUODENUM

Ulcers occur in the duodenum of the same nature and under conditions like those that affect the stomach, but less frequently. They are ten times more common in men than in women; and the ulcers are generally near the pylorus. They cause symptoms similar to those of gastric ulcer, give rise to hæmatemesis, melæna, and perforation, contract adhesions to surrounding parts, or cicatrise and obstruct the duodenum so as to cause dilatation of the stomach. But there are differences. They are often latent: or the early symptoms are weight, oppression, flatulence, and distension after food. When definite pain occurs, it is situated in the epigastrium, or near the right costal margin; and frequently comes on two, three, or four hours after food; or wakes the patient at two in the morning. The pain is often relieved by taking some food. Thus it is due to the acid chyme being passed in the late stage of digestion into the duodenum and over the ulcerated surface: and the introduction of food again postpones this process for a time.

Vomiting is infrequent and does not relieve the pain. Hæmatemesis is much less frequent than in gastric ulcer, but occurs in about one-third of the cases. Melæna sometimes occurs without hæmatemesis; and, according to some, occult blood is very frequent. The appetite is often good. In later stages dilatation of the stomach may be recognised by the usual signs, and, as a rule, no tumour can be felt. If perforation takes place, the symptoms may be less acute, and the collapse less than in gastric ulcer, because the duodenum is often empty, or at any rate contains but little. The escaped fluids tend to run behind the colon, in front of the kidney, into the right iliac fossa, where they may form an abscess; but a subphrenic abscess or an acute general peritonitis is of course possible.

Diagnosis.—This must be made by the situation and character and time of the pain: and its occurrence in attacks, with intervals of comparative freedom. It may be confounded with *hyperchlorhydria*, in which pain comes late and is relieved by solid food: but the secretions are often strongly acid in duodenal ulcer. The occurrence of melæna assists the diagnosis; and occult blood should be looked for. Einhorn uses a "thread test" in this way: a "duodenal bucket" enclosed in a gelatine capsule attached to a white silk cord or thread is swallowed by the patient, and allowed to go down for a length of 75 centimetres (30 inches) from the teeth. At 7 or 8 A.M. it is removed, and if the thread has been in contact with an ulcerated surface, it is stained brown or dirty black for 1 or 2 cm. at a point, from the position of which the site of the ulcer may be inferred. A duodenal ulcer stains the thread at from 58 to 66 cm. (23 to 26 inches) from the teeth. The possibility of *gall-stones*, and of *chronic appendicitis*, must always be considered: they may cause pain and tenderness in the same situation, and in nearly the same circumstances, as does duodenal ulcer.

Treatment.—The duodenal ulcer may be treated in the same way as the gastric ulcer. It is perhaps less amenable, and may sooner call for surgical methods.

CANCER OF THE STOMACH

Ætiology.—Cancer of the stomach is rarely seen before the age of thirty, and the majority of cases occur between forty and sixty. Sex has no appreciable influence; and though there can be little doubt that cancer in general is to a certain extent hereditary, this is not a prominent feature of cancer of the stomach. It is equally frequent among the rich and the poor, and is not related to any particular occupation. It frequently happens in those who have been hitherto quite healthy, and is not determined by any previous disease of the stomach, with the exception of ulcer, which undoubtedly sometimes terminates in cancer: but there is much difference of opinion as to the frequency with which this occurs.

Pathological Anatomy.—Cancer affects all parts of the stomach, but in the majority of cases the pylorus is involved, and the disease extends thence to the adjacent parts of the organs; especially along the lesser curvature. If it affects the cardiac end, the œsophagus is generally also invaded. Sometimes the wall of the stomach is uniformly infiltrated and thickened, and the organ, as a whole, is contracted to a small size. With few exceptions cancer of the stomach is in the form of spheroidal carcinoma, or cylindrical carcinoma; and the former is much more common. Either variety may be scirrhous from excess of fibrous tissue, or medullary from deficiency of it; and colloid degeneration may take place in either, but is more common in the spheroidal variety. The scirrhous change is the most common; squamous epithelioma and sarcoma are comparatively rare.

The cancer commonly begins as an overgrowth of the epithelial cells of the glands of the mucous membrane; the growths project into the submucous tissue, proliferate further, and gradually involve all the coats. In cancer of the pylorus the whole wall of the stomach at this spot is thickened, and projects internally so as to narrow considerably the passage from the stomach into the duodenum; this may admit with difficulty the little finger or a large catheter. The projection terminates abruptly towards the duodenum, more gradually towards the stomach. The thickening mostly affects the submucous layer, but also the muscular coat; and the bands of muscular fibre are separated from one another by the new growth. Subsequently the subserous layer is involved, and deposits of cancer may occur in the adjacent peritoneal surface; in later stages it often ulcerates upon the inner surface. The adjacent mucous membrane may show nodular growths or villous processes.

Important changes occur in the stomach and adjacent parts as a result of cancer, which are in many ways similar to those following simple ulcer. Thus, the ulcerative process may erode vessels and lead to hæmorrhage; this is much less often profuse than it is in simple ulcer. Dilatation of the stomach is very frequent as a result of the narrowing or *stenosis* of the pylorus, which the growth of cancer necessitates; but in a certain number of cases the stomach is actually smaller, and this generally when the cancer affects the whole of the organ, as already shown. *Adhesion* of the stomach to other organs commonly takes place, as the growth reaches the peritoneal surface, and invasion of the organ with cancer may follow. The liver and pancreas are thus frequently attacked; occasionally the spleen or colon. In the last case a *gastro-colic fistula* may result. When the cancer is in front the abdominal wall may become adherent, or, in the absence of adhesion, perforation into the peritoneum may take place; but this is much less likely than in simple ulcer. More often subacute or chronic *peritonitis* takes place without perforation, either spreading from the original lesion, or following a general growth of cancer in the peritoneum. Cancer of the cardiac extremity frequently invades and obstructs the œsophagus.

Secondary deposits occur in various organs, in the peritoneum, as just stated, in the liver, pancreas, and adjacent lymph-glands. These are the mesenteric, retroperitoneal, and portal glands; but, as occurs also in œsophageal cancer, the cervical lymph-glands are sometimes affected quite early. The spleen is more rarely affected; and occasionally more distant organs, as the brain and lungs. After death from cancer, the heart is commonly in the condition known as brown atrophy.

Symptoms.—In the earlier stages there is nothing characteristic about the symptoms, which are chiefly those of dyspepsia. There are discomfort, fulness, weight or pain after food, and acid eructations or flatulence. The *pain* may be at the epigastrium, or in the position of heartburn; small quantities of food may be regurgitated. After a time *vomiting* takes place, at first only at long intervals, then weekly, or two or three times a week.

Pain then becomes a more prominent symptom, and though, like the other symptoms, at first related more or less closely to the ingestion of food, it soon becomes more constant, or arises independently of a meal. It commonly radiates from the epigastric region, where it is, in later stages, most intense, and is occasionally felt between the shoulders, or in the lumbar region. It is often stabbing, and lancinating, but may be boring, burning, gnawing, or tearing.

The vomited matters consist of food in different stages of digestion, mixed with more or less mucus, or streaks of blood. Often the blood mixed with the vomit has the appearance of coffee-grounds (*see* p. 706). Abundant hæmorrhages are much less common than in ulcers; but a large vessel, such as the splenic artery, is sometimes eroded, and profuse and fatal bleeding results.

In the majority of cases, a *tumour* is discovered at some time or other; but rarely in the first three or four months. The position of course varies with the part of the stomach affected. A pyloric tumour is commonly situated in the middle line, or a little to the right, midway between the ensiform appendix and the umbilicus, but it may be close to the umbilicus, or more to the right of the middle line; indeed, almost anywhere in the triangle formed by the right costal margin, the middle line, and a horizontal line running through the umbilicus. When the stomach is much dilated the tumour is even below the umbilicus. It varies in size from that of a walnut to that of a small orange, is generally very hard, sometimes globular, but often somewhat square, and mostly irregularly nodular. It is at first freely movable, and descends on inspiration, but in later stages it may contract adhesions and become more fixed. It frequently receives an impulse from the underlying aorta. On percussion it is dull or imperfectly resonant; handling it causes pain, which may last for some time afterwards.

The condition of the abdomen varies. As a result of the small quantity of food that passes the pylorus, it is frequently empty, but presents in many cases the prominence in the upper or left portion which is due to dilatation of the stomach. Dilatation is further

recognised by the peristaltic wave of contraction, the varying resonance and dulness on percussion according to the amount of the contents, the splashing sounds heard on movement, and the characters of the vomited matter (*see* p. 701).

In some cases the amount of fibroid change in the pylorus is so great that the lesion has been regarded as purely fibrous, and has been called *hypertrophic stenosis*. But the invasion of the lymphatics by cancer shows the nature of the thickening. However, the progress of these cases is slow, and the patients for years suffer only from the resulting dilatation.

Though the pylorus is very frequently the seat of gastric cancer, it must not be forgotten that it occurs in other situations, and that there will be some differences in the symptoms and physical signs in accordance with these. Thus, a tumour in the back of the stomach may grow to a large size without being felt; or it may be felt, but is mistaken for a kidney. The signs yielded by a general infiltration of the gastric walls are very obscure. In all these cases dilatation is not present as it is in so many cases of pyloric cancer. The pain may be also rather in the left loin or back than in the epigastrium.

Indications of severe constitutional disturbance set in comparatively early in cancer of the stomach. The appetite diminishes, the patient loses flesh, strength, and colour; and in advanced conditions *emaciation* and *anæmia* are extreme. The leucocytosis which is natural after meals is often absent in cancer of the stomach. The tongue is often clean, but in the last days may be dry and covered with sordes or deposits of thrush. The bowels are mostly constipated, especially if dilatation supervenes.

Various complications may arise towards the end. Ascites may follow the spread of cancer to the peritoneum, or the implication of the liver and portal vein. Occasionally perforation into the peritoneal cavity takes place, and is followed by peritonitis; but the symptoms of this event are often obscure or not distinctive. Cancer of the retroperitoneal glands causes œdema of the feet; or the same is brought about by thrombosis of the large veins.

Gastro-colic fistula, which is mostly the result of cancer spreading from the stomach to the colon, is marked either by *lienteria*, undigested contents of the stomach passing directly into the colon, and hence *per rectum;* or by fæcal vomiting, the contents of the colon passing into the stomach and hence being vomited. The former is more frequently the case when the pylorus is obstructed.

Death commonly takes place from exhaustion, as a result of continued pain, vomiting, and deficient assimilation of nutriment: or of a rapid spread of secondary growths in the liver with continued pyrexia. Rarely a profuse hæmorrhage, or peritonitis, bronchitis, or pneumonia may terminate the scene.

Duration.—The illness commonly lasts from six months to two years; two-thirds of the cases last less than eighteen months, and a very small proportion more than two years.

Diagnosis.—Cancer is usually distinguished from the majority of the diseases of the stomach by the presence of a tumour. This is, however, not generally felt in the earliest stages, and may be imperceptible later from its small size; from the pylorus lying under the liver; from its being concealed by much distension of the bowels, or by ascites; or finally, because the tumour is situate on the posterior wall of the stomach. Occasionally in such cases the diagnosis has been determined by the discovery of a hard enlarged cervical gland. If no tumour is discoverable cancer may be confounded with chronic gastritis, or ulcer of the stomach; or purely neuralgic pains may be thought to be due to cancer. In *gastritis* the disease may have originated in imprudent diet; the pain and vomiting are more or less intimately related to diet; the appetite is often good, or even excessive; the tongue is furred, and headache, malaise, &c., are present. Cancer, on the other hand, arises independently of previous gastric troubles, and the pain and vomiting are less dependent on food: finally, indeed, the pain becomes continuous. The tongue may remain clean, but appetite is soon lost, and the nutrition of the patient suffers. In gastritis also judicious treatment materially or entirely relieves the symptoms, which in cancer are but little, or only for a time, influenced.

The same important difference in the result of treatment is to be noted between *ulcer* and cancer; ulcer is nearly always improved by proper dietetic treatment, whereas cancer may be scarcely at all relieved. Ulcer also has more localised pain, and the pain and vomiting are aggravated or brought on by food. Profuse hæmorrhage is much more probably the result of ulcer, and often occurs early; in cancer it appears late, if it all; coffee-ground vomit is seen in both. Free hydrochloric acid is rarely present in the vomit in cancer. Anæmia is mostly the result of hæmorrhage in ulcer, but in cancer it develops when the bleeding has been slight or absent. A cicatrised ulcer may lead to troublesome pains, but there is a long history, and the strength and health are fairly maintained. Dilatation of the stomach occurs very late in ulcer, but much more rapidly in cancer. Exceptionally the tumour of cancer may be simulated by the matting and adhesion of parts caused by ulcer (*see* p. 705). The age to which cancer is almost strictly limited and the short duration of the disease, are also important elements in its diagnosis.

In purely *nervous* affections of the stomach the pain is continuous, but there is generally no wasting.

Occasionally the anæmia of cancer has been so marked, and the local symptoms so slight, as to have led to the suspicion of *pernicious anæmia;* this is especially likely where the tumour is small or not easily reached. In such cases repeated examinations of the abdomen must be made, with precautions previously indicated (*see* p. 671).

The absence of free hydrochloric acid from the contents of the stomach is not peculiar to cancer, but its presence on repeated examinations is an argument against that disease. Lactic, butyric,

and acetic acids are likely to be present from dilatation, and fermentation of the contents.

Prognosis.—This is very unfavourable, as death inevitably ensues, unless the growth and all infected glands can be entirely removed. Life may be prolonged by operations, such as gastro-jejunostomy, which obviate the obstructive effects of the growth. But it often happens that by the time the tumour is first felt, and when the symptoms are scarcely enough to alarm, the lymphatic glands have become deeply involved, and the chance of benefit from operation no longer exists.

Treatment.—The first consideration is whether any such operation can be usefully undertaken. Those which may have to be considered are: *Pylorectomy,* or resection of the pylorus; *Gastro-enterostomy; Enterostomy* or *Jejunostomy,* by which a portion of the small intestine is attached to the abdominal surface, and the patient is fed through a fistula made in it. The entire stomach has been successfully removed, and the œsophagus fixed to the small intestine; *œsophago-enterostomy.* For fibrous stenosis of the pylorus, Loreta's operation may be done.

If the position or extent of the lesion renders operation inadvisable, a palliative course of treatment must be adopted. The diet must be arranged on somewhat different principles from those followed in ulcer. Since the symptoms are not solely dependent on food, and the tendency to exhaustion is so certain and pronounced, we must avoid the treatment by abstinence, which is justifiable in the more curable disease. Only rarely, when the pain is very severe, may the patient be fed by enema for a few days; but generally the food must be of a light, nutritious kind, and must be given in the natural way—by the mouth. Milk, milk and soda-water, or milk peptonised with liquor pancreaticus, may be given in some cases; in others it may be thickened with farinaceous food or eggs; or more solid nutriment may be borne, such as fish, chicken, or oysters. Wine of a light kind may be allowed; champagne is often useful on account of its effervescence, and in late stages brandy may be necessary. The symptoms of pain and vomiting must be dealt with much as in ulcer. For vomiting, ice internally, either alone or with milk, and ice applications to the epigastrium; effervescing medicines, small quantities of iced champagne, extract of opium in doses of $\frac{1}{6}$ to $\frac{1}{4}$ grain, or morphia in pill or solution ($\frac{1}{8}$ to $\frac{1}{6}$ grain) should be tried. Opium or morphia is again constantly required for the pain; or locally a small blister or chloroform liniment will sometimes give relief. Constipation must be met by enemata, or by saline purgatives, or by pills of aloes and iron.

If dilatation is a prominent symptom, and large quantities of food are vomited every few days, relief may be temporarily afforded by washing out the stomach daily with the stomach-pump or syphon-tube (*see* Dilatation).

BENIGN TUMOURS OF THE STOMACH

These include *adenoma, myoma, lipoma, fibro-lipoma, lymphadenoma,* and *cysis.* They are quite infrequent. The first two are the more common: and they occasionally cause symptoms by obstructing the pylorus. In other cases the symptoms will depend on the size and position of the growth.

CONGENITAL HYPERTROPHIC STENOSIS

This is a form of pyloric obstruction of which the symptoms appear as a rule from a few days to six or seven weeks after birth. They consist of vomiting, constipation, emaciation, and the vomited matters are often abundant, and thrown up with much force. On careful examination, the peristaltic movement from left to right, so characteristic of gastric dilatation, will be seen; and in nearly all cases a tumour or thickening, one-half or three-quarters of an inch in diameter, varying in consistency, will be found to the right of the middle line, a little below the costal margin. Upon these two features, peristalsis and tumour, the diagnosis depends.

This thickening is a hyperplasia of the muscular fibres of the pylorus, chiefly of the circular coat, and probably developed during fœtal life: within the thickened mass, the mucous membrane is thrown into folds.

Treatment.—Many cases have been treated surgically by pyloroplasty, dilatation, and gastro-enterostomy; but some have recovered with simple dieting, very small quantities (from a teaspoonful to two or three tablespoonfuls) of whey, milk and water, peptonised milk, albumen-water, or meat-juice, being given at intervals of half an hour or longer. In these cases lavage, once or twice daily, has also been of great service. If, after some days of this treatment, the child continues to vomit or to lose weight, an operation should be performed.

DISEASES OF THE INTESTINE

CONSTIPATION

The healthy action of the bowels depends on a sufficient supply of food, the waste of which forms the material for the fæces; a natural secretion of intestinal juices; and an intestinal muscular system readily stimulated and strong enough to force on the fæces from point to point. This action, however, varies in different individuals, who may still all be healthy. Most persons have an action of the bowels once a day, but others twice a day, and some only every other day.

Constipation is the retention of fæces for longer than the normal

period of twenty-four hours, or in some persons two days; and is due to delay either in the general movement along the large intestine, or in the evacuation of the pelvic colon and rectum, or in both the processes.

Hertz, using the Röntgen rays upon persons fed with bismuth meals (*see* p. 687), shows that the average times for food to reach the different parts of the intestinal canal are as follows:

Cæcum, 4½ hours; hepatic flexure of colon, 6½ hours; splenic flexure, 9 hours; commencement of iliac colon, 11 hours; pelvic colon, 12 hours; rectum, 18 hours.

The desire to defæcate is caused by the entry into the rectum of the fæces which have accumulated in the pelvic colon during twenty-four hours, and this is brought about by the stimulus of eating breakfast, or of getting up, or of some other daily recurring function.

Constipation may be occasional, and due to accidental causes outside the alimentary functions, or it is frequently repeated and constant, the so-called "habitual constipation."

The general movement of fæces along the intestinal canal is purely involuntary, and is dependent upon the adequate muscular power of the intestinal wall, properly stimulated to action by suitable food. A weak musculature may be a hereditary failing, or it may supervene in later life as a cause of senile constipation. A temporary weakness occurs in fevers, and acute illnesses, and may result from anæmia, chlorosis, rickets, and diseases involving nervous depression, such as melancholia, neurasthenia, &c. It is often present in cases of cerebral tumour. Locally it may be due to flatulent distension, and to catarrh of the mucous membrane.

The stimulus to the bowel comes chiefly from the food; and this may be insufficient in quantity, or too dry, or deficient in mechanical stimuli, of which the cellulose of vegetable substances is the most important. It appears, also, that in some individuals, unusual powers of digestion and absorption on the part of the intestines, may leave so little residue, that evacuations must be infrequent. In many gastric disorders especially where vomiting is frequent, constipation occurs. Further, the reflex action of the bowel may be directly inhibited by painful local affections of an inflammatory or traumatic nature in the pelvis or abdomen.

The defect in the final process of defæcation, that is, in the passage of fæces into the rectum, and the final evacuation, is the cause of the larger proportion of cases of so-called habitual constipation. It is called by Hertz *dyschezia* (χέζω, to ease oneself). The performance of this function is dependent upon a stimulus conveyed from the rectum, and the response upon the part of the pelvic colon. In well-regulated persons, the stimulus arises at a given time every day, and if allowed to operate an evacuation is the result. If the stimulus is disregarded, and the desire to go to stool is repressed, the reflex is likely to be less active on a subsequent occasion, and in course of time the stimulus

may fail to be felt. Thus, the repression of the desire and the disregard of the sensation are common causes of constipation. Want of time in the case of persons hurrying to business, false modesty in large houses, or in girls' schools, inadequate supply of accommodation in large establishments, mere laziness in many people, contribute to this, and lead to a postponement of the process until the regular habit is entirely given up, the fæces are retained for two or three days or even a week, and then evacuation can only be secured by the use of aperient drugs, or an enema.

Another cause of dyschezia is weakness of the voluntary muscles which compress the abdominal contents, and thus assist in the passage of fæces from the colon into the rectum, and from the rectum through the anal passage. These are the abdominal expiratory muscles, the diaphragm, and the levator ani and other muscles of the pelvic floor.

In any part of the large intestine delay may be caused by mechanical obstacles to the passage of the fæces, such as compression or kinking of the colon, hard fæcal masses, stricture by growth, a retroverted uterus, spasm of the sphincter ani, and spasm of the colon, a condition described as *enterospasm* (*see* p. 753). Some of these conditions in a higher degree lead to complete obstruction.

Symptoms.—If left to themselves the bowels only act at intervals of two, three, four, or more days; the rectum becomes loaded with hard round masses of fæcal matter (scybala), generally rather pale, which are welded together into masses. The desire to go to stool perhaps at first only results in ineffectual straining efforts; but finally some scybala are passed, and the same may be repeated two or three times within a few hours, till the lower bowel is emptied. After this the bowel is inactive for another period of several days. During the retention the patient may suffer various inconveniences. Locally, there may be a sense of fulness in the perineum, or pruritus ani; and the hæmorrhoidal veins may swell and possibly varicocele may be caused. Sometimes there is pain down the thigh from pressure of the fæcal masses on the nerves in the pelvis; moderate distension of the abdomen often occurs, with perhaps flatulence and eructations, the tongue is often furred, whitish or dirty brown, and the breath may be foul. Some patients feel languid, confused, wanting in vigour or freshness, and have actual headache, or even a great deal of mental depression. But it must be noted that often the more habitual the constipation, the less is the general disturbance; and many are not conscious of having anything the matter with them, though their last evacuation was many days before.

It is generally believed that, normally, when the fæces have passed out of the pelvic colon they are promptly discharged from the anus without any lengthened stay in the rectum; but it is certain that in habitual constipation, either from loss of sensitiveness or lessened power, the rectum tolerates the presence of fæces and may become enormously dilated to accommodate them. The scybalous condition

of the fæces is explained by their retention in the pelvic colon, during which there is time for the absorption of most of the liquid contained in them. Even when the rectum is distended with scybalous masses, some fæcal fluid may escape from the anus, or a secretion of mucus may be excited, and the discharge of these liquids may simulate a diarrhœa. A more extensive catarrhal colitis, and stercoral ulcers, may also result from constipation.

The accumulation of fæcal matter in the pelvic colon may be such that it forms a large tumour in the lower part of the abdomen, of which the distinguishing feature is the fact that it can be indented by firm pressure with the finger.

Treatment.—For many cases of troublesome constipation much may be done without having recourse to drugs. The patient should make a regular daily visit to the closet, whether he feels any desire or not at the time; and this should be continued as a habit for the rest of life, but it may take months before its good effects come into full operation. The diet should be modified so as to include a sufficiency of vegetables, fruit, fresh or preserved, or salad with salad oil; brown bread, wholemeal bread, or oatmeal porridge sometimes supplies the desired stimulus to the bowel. The diet, also, should be liquid enough; and with some a daily evacuation is ensured by drinking a tumblerful of cold water or eating an apple before breakfast. To those of sedentary habits, walking exercise, fencing, horse-riding or driving is often of benefit, or the abdominal muscles may be specially exercised by gymnastics.

But with all this it may be still necessary to have recourse to drugs, and a careful selection is requisite. As a rule, very active or drastic purgatives must be avoided; they produce abundant liquid motions, from the effect of which the intestinal muscle is completely exhausted, and consequently no further evacuation takes place for days afterwards. But it has already been shown that constipation depends on weakness of peristaltic action, and hence over-stimulation and exhaustion are especially to be avoided. From this point of view much advantage is gained by combining with the ordinary laxatives such drugs as have a tonic effect upon the intestinal muscle. These are especially nux vomica and iron.

The remedies more commonly employed are the saline mineral waters, as Friedrichshall, Püllna, Hunyádi János (containing the sulphates of magnesium and sodium), and Carlsbad (mainly sulphate of sodium); the patient may take from a wineglassful to half a tumblerful before breakfast. Carlsbad salts extracted from the water of the different springs, of which the Sprudel seems the most efficient, may also be given—a teaspoonful is dissolved in half a tumblerful of hot water, and drunk before breakfast. Another useful laxative is the cascara sagrada: it is less likely than some other vegetable laxatives to exhaust the bowel; it may be given every night in doses of 30 or 40 minims of the liquid extract combined with syrup of ginger, or two or three grains of the solid extract in pill. An efficient combination is that of aloin with

extract of nux vomica, one grain or a grain and a half of the former with a quarter or half a grain of the latter, given in the morning before breakfast; a quarter of a grain of extract of belladonna, or half a grain of ipecacuanha is sometimes usefully added. Sulphate of iron (one grain) with aloin and nux vomica is also very useful. If a daily pill is insufficient, two or even three pills may be taken; but in either case the essence of the treatment is that active purgation should be avoided, and directly this seems likely to be produced, the three pills each day must be reduced to two, or the two to one; and ultimately the bowels will act without any assistance whatever. Galvanism, massage, and kneading of the abdomen are means which may be resorted to in exceptional cases. Enemata of cold water are often necessary when the delay is in the rectum, which is below the point of direct operation of aperients. When a large accumulation of fæces has taken place, the enema may be the only means of clearing the rectum, and it may have to be assisted by the use of the finger: for some days after this the enema may be used to supply the stimulus until a more natural method is established. For an occasional evacuation the injection into the rectum of a drachm of glycerine is often useful.

If adhesions are recognised as a cause, they may be divided.

DIARRHŒA

By diarrhœa is meant the passage of motions more often, and of looser consistence than is normal. This is a frequent result of inflammation of the bowel or enteritis, under which head it will be mentioned; but it is also set up by excessive peristaltic action, and by increase of the intestinal secretions, and there are cases in which it is not easy to say whether a catarrh of the bowels has any share in its production or not.

Causation.—The more common causes are irritating and toxic food, whether solid or liquid, and impurities in drinking-water, or in the respired air. The production of diarrhœa is also illustrated by the use of purgative and laxative medicines, some of which act by exciting the muscular fibre, others by stimulating the intestinal glands, and others by actually inflaming the coats of the bowel. Besides the catarrhal forms of enteritis, there are other changes in the bowel, which are accompanied by diarrhœa. Such are typhoid and tubercular ulcerations, which affect the lower end of the ileum, and perhaps the cæcum; dysentery, already described, which affects the colon and cæcum and sometimes the lowest part of the ileum; lardaceous disease, which invades both small and large intestines in cases of chronic suppuration and chronic syphilis, but most frequently in phthisis, where it is associated often with tubercular ulceration. Another condition of the bowel, which is accompanied with diarrhœa, is *lympho-sarcoma*, which grows in the walls of the intestine, and there may attain a thickness of half or

three-quarters of an inch, while the bowel itself, instead of being contracted by the growth, may be actually enlarged to a circumference of ten or twelve inches. It is of very rare occurrence.

Diarrhœa also occurs in some general toxic conditions, such as septicæmia and uræmia; and occasionally it accompanies the termination of a pneumonia or other acute fever by crisis (*critical diarrhœa*). Some forms of diarrhœa are traceable to a disturbance of the nervous system, such as the emotion of fear, or in some persons almost any form of emotion. A purely hysterical diarrhœa may occur, and the diarrhœa which is an occasional symptom in Graves' disease seems to be related to the other nervous phenomena.

It must be remembered that the frequent discharge of liquids in small quantities does not of itself show that the canal of the bowel is free; thus intussusception which partly obstructs the gut is accompanied by the passage of mucus and blood; fæcal fluid mixed with mucus may find a way past very large masses of impacted fæces; and lastly, even a distinctly contracted intestine may allow some of the thin liquid which collects above the obstruction to pass through and simulate a diarrhœa.

Varieties.—Diarrhœa has received different names according to the nature of the matters passed: thus we have *choleraic* diarrhœa, in which the stools are profuse and watery, or like the rice-water stools of cholera; *dysenteric* diarrhœa, in which mucus is largely present, and perhaps blood; *lienteric* diarrhœa, or *lienteria*, the passage of undigested food; and *bilious* diarrhœa, where the discharges are deeply stained of a brown or greenish-brown colour, which is due, not so much to any increase in the quantity of bile secreted, as to the fact that the contents of the duodenum and jejunum stained with bile have been hurried through the alimentary canal, without giving time for the natural reabsorption of the altered bile-pigment (urobilin). *Colliquative* diarrhœa is a term applied to the profuse, exhausting, and intractable discharges which occur in the last stages of phthisis.

Treatment.—This must depend upon the cause, or the associated condition. A critical diarrhœa may generally be left to itself, and some caution must be exercised in checking those which result from the congestive catarrh of heart and lung disease, or take place in Bright's disease. The treatment of the diarrhœa of typhoid fever has been described (*see* p. 54). In most cases, not of a specific nature, the treatment described under Enteritis may be employed. The rapid action of the bowels immediately upon taking food, which sometimes results in lienteria, and which seems to depend upon an exaggerated intestinal reflex, may be treated with full doses of potassium bromide

HÆMORRHAGE FROM THE BOWEL

The passage of blood *per rectum* has already been noticed as occurring in enteric fever, and in ulcer of the stomach. It also results from other ulcerations, as ulcer of the duodenum, dysentery, and ulcerative colitis; from intussusception, from cancer of the sigmoid or rectum, from conditions of intense congestion, from embolism or thrombosis of mesenteric vessels, and from purpura and other conditions of blood-disease. The way in which the blood is passed may give a clue as to the point whence it comes. In bleeding from gastric or duodenal ulcers the blood is considerably altered by the secretions, and forms a black, tarry, semi-liquid or treacly mass (*melæna*); in hæmorrhage from typhoid ulcers the blood is equally unmixed with fæces, but brighter red and more fluid than in the former case, from the action of the alkaline contents; the blood in dysentery is in streaks or small clots mixed up with mucus or pus, or thin fæcal matter, though from time to time small quantities of pure blood may be passed. Large quantities of blood may be lost from piles, or from an ulcer of the rectum. Here the bleeding is generally caused by the act of defæcation, the blood either streaking one side of the solid fæcal mass, or coming more or less pure in drops or streams after the motion is evacuated. In scorbutic, purpuric, and hæmorrhagic conditions (scurvy, purpura hæmorrhagica, acute yellow atrophy of the liver, malignant variola) blood comes from the rectum more or less mixed with fæces, or pure, according to the part of the intestine yielding it, or the freedom with which it escapes. The **Treatment** of hæmorrhage is described with the various diseases which may cause it.

INTESTINAL COLIC

The term *colic*, though obviously derived from the word *colon*, means a spasmodic abdominal pain, or a pain presumably due to contraction of visceral muscular fibres. These fibres may be those of the ureter (renal colic), of the bile ducts (biliary or hepatic colic), or of the intestines (intestinal colic).

Ætiology.—The most frequent cause of intestinal colic is irritating and unsuitable ingesta, such as pork, cheese, high game, shell-fish, ices, &c. In children, colic is a common result of indigestible food, or even simple excess. With these may be classed the more active purgatives. On the other hand, constipation is often associated with colic, and this is markedly so in the colic due to lead poisoning, whether acute or chronic (*see* Lead Poisoning). Some cases may perhaps be referred to a purely nervous source; for instance, the severe pain of gastric crisis in locomotor ataxy. Lastly, mechanical and acute inflammatory lesions of the bowel, such as strangulation and intussusception, lead to severe pains,

which are partly or wholly due to muscular contraction. The term *colic* is, however, generally reserved for conditions in which there is no structural or inflammatory change.

Symptoms.—The important symptom is pain, which is situate about the umbilicus, but may move about other parts of the abdomen. This pain is often relieved by pressure, but sometimes there is tenderness. The abdomen is either drawn in, and the abdominal muscles are contracted, or the belly is distended from the presence of flatus. When flatus is present borborygmi are produced by its movements, as it is driven on by the varying intestinal spasm.

The pain may be so severe as to cause much collapse, with profuse clammy sweat and small feeble pulse. Sometimes there is vomiting; often there is constipation; on the other hand, some ingesta, which cause colic, set up active diarrhœa with brown watery stools, and mucus after a time. Here the colic is associated with a definite though slight enteritis. The more active purgatives also produce griping and "colicky" pains, which are commonly diminished after each evacuation.

Diagnosis.—Gastric and intestinal colic may be confounded with the pains of any acute inflammation in the abdomen, such as appendicitis, peritonitis, pancreatitis, cholecystitis, with the pain of acute strangulation of the intestine, and with hepatic or even renal colic; the special features of these several diseases must always be carefully considered. Lead colic is often mistaken for intestinal obstruction, when a glance at the gums would give the right clue. On the whole, it is the absence of the more positive indications of obstruction, peritonitis, or other acute inflammation, and of the facial signs of abdominal disease, the relation to unsuitable foods, or the fact that the patient has had similar attacks before, which will help most to make the case clear.

Treatment.—Obviously, cases of severe abdominal pain must be treated with much caution. If the pain is certainly due to irritating ingesta, relief generally follows the exhibition of purgatives, such as an ounce of castor-oil, with 15 minims of tr. opii, or half an ounce of magnesium sulphate with half a drachm of tr. hyoscyami, or 5 grains of calomel; and a similar line of treatment is used in lead colic (*see* Lead Poisoning). A warm-water or castor-oil enema may also help; and hot fomentations or a hot-water tin should be applied to the abdomen. If there is any likelihood that peritonitis or obstruction may be present, it is better to treat the case with anodynes, such as one grain of opium internally, repeated in two or three hours, or the subcutaneous injection of morphine; while the external applications are also used. The successful result, if it is colic, will not be materially delayed; while the injury that a purgative may do is avoided.

ENTERALGIA

This term is the equivalent of intestinal neuralgia. In cases included under this head there are attacks of severe abdominal pain, mostly near the umbilicus, not related to food, without vomiting, distension, or other indication of organic disease. Phenazone and bromides may be given during the attack, and tonics in the intervals.

ENTERITIS

There are several conditions, affecting different parts of the alimentary canal, which may properly be termed enteritis, or inflammation of the intestine. For instance, the catarrhal process, of which some forms of diarrhœa are the result; tubercular and typhoid ulcerations in the ileum; the ulcerative inflammation of the colon, known as dysentery; and the acute changes set up by intussusception and strangulations, are all, in fact, enteritis. But a large number of these have already received distinctive names; others are only secondary conditions, which produce few symptoms beyond those of the primary disorder; and in others, again, inflammation of the coats of the intestine involves a peritonitis, which throws the symptoms due to the mucous inflammation completely into the shade. Thus the number of cases which require separate description as enteritis is but a small one, though it is probable that a fair consideration of the pathological side of many of our intestinal cases, such as diarrhœa, would show that the name might be justly used more often.

The following forms of enteritis will be here described: *Catarrhal enteritis, infantile enteritis, food-poisoning, sprue, diphtheritic enteritis,* and *phlegmonous enteritis*

CATARRHAL ENTERITIS

(*Intestinal Catarrh*)

Anything which irritates the mucous membrane of the intestine may set up catarrh—such as unsuitable food, certain poisons, and purgative drugs. It is also ascribed sometimes to chill; but a much more potent factor in its production is excessive heat, and it is always more prevalent in the hot weather of summer and autumn than in the remaining part of the year. This frequency in the summer concerns people of all ages, but infants are especially attacked. In very hot weather every kind of food decomposes more readily, and milk quickly "turns": and these processes are no doubt dependent upon micro-organisms. An important factor related to summer heat is the prevalence of flies, which may convey infection to both meat and milk, not properly protected from them. Many kinds of organisms have been found in the intestines in health and disease; those most constantly present are the *bacterium lactis*

aërogenes in the upper part, and the *bacillus coli communis* in the lower Catarrh of the intestines may also arise from passive congestion in cardiac and hepatic disease.

Anatomy.—The changes in the mucous membrane of the intestine are similar to those in other parts of the body. The tissues are more vascular, and become swollen, and large quantities of mucus are excreted. The epithelial cells of Lieberkühn's glands are swollen, cloudy, and become detached, and cellular infiltration takes place in the intertubular tissue. In more advanced cases the solitary follicles are enlarged, and they may become eroded, and produce small ulcers (*follicular enteritis*). In some cases, also, ulcerations occur in other parts of the mucous membrane, and the secretions may consist of muco-pus, or even pus. As a rule, the inflammation subsides, but it may relapse into a *chronic* condition, with more prominent changes in the mucous membrane. Sometimes there is considerable thickening, with slaty discoloration of the surface; often—especially in the chronic catarrh of infants—there is atrophy of the mucous membrane, involving the glandular layer, but leaving the muscular layer of the mucous membrane, and the submucous tissue, intact.

Symptoms.—The chief symptom of enteritis is diarrhœa, or the frequent passage of motions, loose or liquid in consistence. This symptom is due, not only to the alteration in the secretions poured into the intestinal canal, but also, and largely, to the increased peristaltic movements which the irritation of the mucous membrane calls forth. The condition of the fæces varies much: they are generally at first abundant, liquid, and brownish in colour, with flakes or lumps of more solid matter; but they soon become paler, or, it may be, yellowish or sometimes green; in consistence they are often quite watery, or perhaps slimy, or they contain lumps of brown mucus. Under the microscope there are particles of undigested food, meat fibre, starch granules, and fat, with crystals of ammonio-magnesian phosphate, epithelial and pus cells, and bacteria. The bowels may be moved from two or three times a day to ten, twelve, or more.

Pain is often present, but not so much continuously as in the form of colicky or griping attacks, which precede the passage of the motions and subside again afterwards. Actual tenderness is not generally present, nor does examination of the abdomen reveal anything characteristic. Gurgling noises and borborygmi from time to time accompany the more active intestinal movements. The temperature is variable: it may be raised one or two degrees, or remain normal. Frequently the appetite is lost, the patient complains of thirst, the mouth is dry, the tongue is slightly furred, and a considerable degree of bodily weakness results when the diarrhœa is excessive. A very sudden and acute attack may begin with vomiting

In most cases the symptoms pass off in the course of a few days; the diarrhœa may cease suddenly, leaving a long interval before

the bowels are again opened, or the motions may gradually become less frequent, gaining a firmer and firmer consistence. If the complaint becomes chronic, the patient is troubled with three or four evacuations daily of watery mucus, with occasional griping pains. The imperfect digestion and absorption of food may lead to considerable loss of nutrition.

The disturbances of enteritis frequently extend to the large intestine, so that strictly an *entero-colitis* results. When they can be discriminated catarrh of the *small* intestine is more likely to be present if the stomach is at the same time involved; it is less likely to be accompanied by diarrhœa, which must depend finally upon the action of the large intestine. The evacuations often contain bile and undigested food; and mucus, if present, is more intimately mixed with the fæces. In catarrh of the *large* intestine, the mucus occurs in separate masses; muco-pus or pus itself may be recognised. As catarrh approaches the *rectum*, tenesmus is more likely to be a symptom.

Treatment.—The patient should remain in bed and be kept warm. Beyond this, in slighter cases, a careful regulation of the diet may be all that is necessary. Gruel, arrowroot, beef-tea, or mutton-broth, with rusk or toast, milk and soda-water, or milk and lime-water in small quantities at a time, should take the place of the ordinary meals. They should not be given too hot. Many cases, however, will require drugs in addition. If it is quite certain that irritating matters, such as unripe fruit, are the cause of the attack, and are still in the bowel, a laxative may be given in order to get rid of them and so prevent further irritation. For this purpose a single dose of castor oil may suffice, or one of compound rhubarb powder or of calomel. But generally, by the time the case comes under treatment, there has been a free evacuation, and it is desirable to check the excessive peristalsis and abundant discharge, as well as to relieve pain. The tincture of opium may be given in doses of 5 minims every four hours, combined with an astringent such as hæmatoxylum, catechu, tannigen (5 gr. in cachet), the aromatic chalk powder, or dilute sulphuric acid. Bismuth carbonate and bismuth salicylate are also of value, and may be given with opium. If the griping is very severe, morphia may be injected subcutaneously. If the diarrhœa is obstinate and exhausting, an enema of two ounces of starch containing 15 minims of laudanum may often be used with success. In children, opium must be used as little as possible; aromatic chalk powder and bismuth carbonate or salicylate must first be tried. If opium be necessary, the dose for a child one year old should not exceed one minim of the tincture, and it is well to begin with less.

In chronic intestinal catarrh, rest, careful regulation of the diet, with opium, astringents and antiseptics, are still required. Here also enemata may be found useful—two or three pints of warm water containing 1 per cent. of boric acid, salicylic acid, tannin, or acetate of lead.

INFANTILE ENTERITIS

Children are subject to a chronic intestinal disorder, which is probably, in part at least, of a catarrhal nature. It is by far most frequent in infants who cannot be brought up at the breast, or must be weaned early and have to depend on artificial feeding. Sometimes immediately after birth, or directly upon weaning, in other cases after a longer or shorter interval, the food disagrees; the child vomits, and the motions are loose; or with not much vomiting, there is constant diarrhœa. If it is taking milk, this is returned in curdled lumps; by the bowels also particles of undigested milk are passed, but the fæces, which in the healthy infant are yellow, generally become grass-green in colour, are slimy from mucus, and very offensive. Among the poorer people, children under twelve or fifteen months are often fed with potato and meat, much the same as their parents, and in such cases undigested particles of these foods are found in the motions. The children suffer from griping pains, which are brought on by the ingestion of food, or occur just before a loose evacuation. Frequently the abdomen is distended with flatus, but presents nothing on examination with the hand; the condition, though often spoken of by the parents as "consumption of the bowels," has no necessary connection with either tubercular ulceration, tubercular peritonitis, or caseation of the mesenteric glands (*tabes mesenterica*). After a certain time the child emaciates, lies fretful in its mother's arms, and is constantly whining, or screaming from time to time with sudden pain. Finally, collapse and death may terminate the scene.

The *summer diarrhœa* of infants (*epidemic infantile diarrhœa*) is a gastro-enteritis, in which the intestinal element is generally the more prominent. It is due in great part to contaminated milk: and this contamination is largely promoted by contagion carried by flies. Possibly also flies carrying bacteria directly infect the infant's lips or mouth. The symptoms come on acutely: and death may result in a few days, preceded by collapse, with depressed fontanelle, and unconsciousness; or the diarrhœa persists for many days very little amenable to treatment.

A not very common condition allied to enteritis is one in which the child passes abundant pale or almost colourless semi-fluid stools, like gruel or porridge, with a very offensive odour. The abdomen is full but not tense, the child emaciates, and death often occurs after some weeks' illness. The disease has been called the *cœliac disease* by Gee, and *acholia* by Cheadle.

Morbid Anatomy.—In most cases the appearances in the intestine are confined to enlargement of the solitary follicles, with perhaps abrasion and ulceration; in old cases the intestines may be atrophied. Both small and large bowel may be involved. Rickets, or tubercular and caseating glands, if present, must be regarded as secondary or independent.

Treatment.—The management of the child's food is the first consideration. If the child is suckled, it should be seen that the mother's milk is of good quality, and that it is not given too frequently; a baby of one or two months old should not be nursed more than once every two or three hours, and the mother must carefully abstain from giving it the breast simply because it cries. If from any cause the mother is unable to suckle it, the most efficient substitute is a wet-nurse, and if this cannot be provided, recourse must be had to the milk of domestic animals—namely, the cow, goat, or ass. Such milks differ from human milk in composition and in the reaction of the casein to the acid of the child's stomach. The average percentage composition of human milk is proteid 2·0, fat 3·5, sugar 6·5, mineral matter ·2, and water 87 or 88. Cows' milk contains twice as much proteid, more salts, only slightly more fat, and less lactose, or sugar of milk: the casein coagulates in large lumps. Of the proteids in human milk, the caseinogen is only half as much as the whey-proteids (chiefly lact-albumin), in cows' milk it is two and a half times as much: thus there are five times as much caseinogen per cent. in cows' milk as in human milk. Goats' milk is very similar to cows' milk, but contains more fat: the casein coagulates in large masses. Asses' milk is deficient in solid constituents, but the casein clots in small particles like that of human milk; it is, therefore, often digested with ease, but it is not sufficiently nutritious to be continued for long. Moreover, the greater facilities for obtaining cows' milk will always be a reason for using it, if it can be so modified as to approximate its composition and properties to that of human milk. The addition of water will reduce the proportion of the proteids and the salts, but the mixture must be sweetened, for instance, by the previous solution of one ounce of milk-sugar in each pint of water; and one drachm of cream (containing 20 per cent. of fat) should be added to each ounce of the milk-and-water mixture, or a drachm each of cream and milk-sugar may be added to every four ounces of water and milk mixed in the following proportions: for infants under one month old there should be one part of pure milk to two parts of water, for those a little older up to four months equal parts of milk and water, and after that age two parts of milk to one of water. The dilution also slightly influences the coagulation of the casein; but this is rendered still more like that of human milk by the addition of lime-water or barley-water. Lime-water, moreover, neutralises the acidity, which distinguishes cows' milk from that of women; it should be in the proportion of one part to two parts of the pure milk employed. Another mode of getting over the difficulty of the digestibility of the casein is to peptonise the milk by means of Benger's liquor pancreaticus or Fairchild's peptogenic powders. This may suffice for a time, but the objection is that it is doing the stomach's work entirely, and depriving it of the exercise of its proper function.

More accurate methods of compounding a suitable milk have been devised, and such an artificial human milk is either a direct

mixture of cows' milk, cream, lactose, and water in certain proportions; or it consists of cows' milk from which some portion of the caseinogen has been removed. Rotch uses a mixture of: Milk, 1 ounce; cream (20 per cent.), diluted with one-third part water, 2 ounces; milk-sugar, $3\frac{3}{8}$ drachms in water, 3 ounces; lime-water, diluted with three-fourth parts water, 2 ounces. Cheadle altered the milk as follows: The cream was removed by skimming, after the milk had stood some time. The remainder was divided into two equal portions, from one of which the caseinogen was removed by rennet. The other half was then added to the whey, and the whole of the cream added. For an older child, the caseinogen was removed from a third part only.

But cows' milk, either in transmission or while standing, may be readily contaminated with organic matters of a bacterial nature. Infection by flies should be prevented by keeping it in covered vessels, or in jugs covered with muslin. Contamination, if it is likely to have occurred, must be corrected by *sterilisation*—that is, either by boiling or steaming the milk, or by pasteurisation, or by refrigeration. Boiling has the disadvantage of coagulating the albumin, which, with some fat, is removed in the scum; and the milk is rendered less palatable. But it is a convenient method, and the loss of nutrient value is slight: the boiling, however, must not be prolonged. In the different forms of *sterilisers* the milk is heated just short of boiling, either by exposure in bottles to steam, or by the immersion of a vessel containing the milk in a larger vessel containing water, which is then boiled. The bottles are loosely plugged while exposed to heat, and tightly stopped when the process is complete. Steaming for twenty minutes will suffice for milk to be used immediately, but if it is required to keep for the day a longer time must be given. Rotch directs that his mixture shall be sterilised before the lime-water is added.

Pasteurisation is the exposure of the milk for fifteen or thirty minutes to temperatures below that of boiling, which are sufficient to check the growth of bacteria, and do not damage the milk as boiling does. These are 65° to 70° C., or 149° to 158° F.

Refrigeration answers the same purpose, since a temperature of 40° F. will entirely stop bacterial growth. The milk should be taken direct from the cow to the refrigerator, and when reduced to 40° F. should be bottled and stored at that temperature.

"Humanised" milk can be obtained which is both sterilised and assimilated to the normal composition. A sterilised milk diminishes the risk of infection by typhoid fever, scarlatina, and diphtheria, when those diseases are prevalent. But its unduly prolonged use has sometimes produced infantile scurvy, and this may have to be provided against (*see* Scorbutus).

The quantities of food required by a bottle-fed child may be stated as follows: In the first week of life an ounce every two hours; in the first month $1\frac{1}{2}$ to 2 ounces; and an ounce may be added to each feed for every month up to the sixth; while the interval between the feeds is gradually increased to three hours in

the fourth month. But some regard must be paid to the size and vigour of the infant.

If the child is fed with the bottle, it is essential that there should be no rubber tube on it, but that the nipple should fit directly on to the glass neck: that it should be kept scrupulously clean in all its parts, and washed thoroughly and scalded after every feeding.

Sometimes it is necessary to stop all milk food for a time, when albumen-water (the white of one egg stirred up in half a pint of water), small quantities of raw meat juice, slightly sweetened (a teaspoonful every four or six hours), or Brand's essence, or diluted Valentine's meat-juice may be supplied instead. In very bad cases of diarrhœa and vomiting, it may be desirable to infuse into the connective tissue normal saline solution, not more than four ounces at a time; or give the same in a small enema, before having recourse to albumen-water and raw meat juice (H. T. Hicks).

Medicinally, very small doses of mercury may be given with advantage—for instance, calomel, $\frac{1}{8}$ or $\frac{1}{6}$ grain with sugar, or hydrarg. c. cret., $\frac{1}{2}$ grain or 1 grain, with 1 or 2 grains of bicarbonate of sodium, two or three times a day. This, with the improved diet, will often be sufficient; but if diarrhœa is obstinate and associated with much pain, it is desirable to give bismuth (2 to 5 grains of the carbonate or salicylate) or astringents, such as aromatic chalk powder (5 grains), extract of hæmatoxylum (2 grains), tannigen (2 grains), or tincture of catechu (10 or 15 minims). Either of these may be combined for older children with small doses of opiates in the form of Dover's powder ($\frac{1}{2}$ or 2 grains) or tincture (1 minim to a child one year old).

If there is much collapse brandy must be given in doses of 10 to 30 minims every three or four hours, or liquor strychninæ ($\frac{1}{2}$ or 1 minim) should be injected subcutaneously.

For the acute forms of summer diarrhœa, a purgative, castor oil or calomel, may be given early; vomiting may be combated by washing out the stomach, and bismuth carbonate with sodium carbonate (3 to 5 grains of each), should be given later.

FOOD-POISONING

It has been already stated that simple catarrhal enteritis is probably due in many cases to micro-organisms, but there is a large class of cases, in which, either from the obvious fact that decomposing food was ingested, or from the occurrence of the illness simultaneously in a number of persons who have all fed from the same, perhaps unsuspected, food, it is quite clear that the food is responsible for the symptoms, which are those of an acute gastro-enteritis. Some of these cases were formerly called *English cholera*, or *Cholera nostras*. More recently they have often been described as instances of *ptomaine-poisoning*, but on very insufficient grounds: for, as a fact, ptomaines are rarely, if ever, found either in the food or in the organs of the sufferers: whereas certain bacteria have been constantly found, and the presence of toxins demonstrated.

The organisms which have been most frequently found are the *Bacillus enteritidis* of Gaertner, *B. paratyphi B.*, *B. proteus*, and *B. coli communis:* the two former in cases due to meat from diseased animals, perhaps slaughtered because they were ill; the two latter in cases due to decomposing meat from healthy animals

In the former group the symptoms come on as a rule within from six to twelve hours after eating the food, and consist of vomiting, diarrhœa, colicky pains, numbness and weakness; with perhaps albuminuria, catarrhal pneumonia, and cutaneous lesions, such as herpes, erythema, urticaria, and petechial hæmorrhages. In the less acute and more prolonged cases there may be a close resemblance to typhoid, or paratyphoid, fever. The cases are sometimes fatal, and the autopsies have shown acute gastro-enteritis, some times with hæmorrhages, swelling of Peyer's patches, enlarged spleen, and congestion of the liver and kidneys. The bacilli may be isolated from the blood, bowels, or solid organs. In the second group of cases, those due to B. proteus and B. coli in decomposed meat (smoked ham, sausages, &c.), the symptoms are similar, but as a rule there is no fever. There are headache, vomiting, dysenteric diarrhœa, weakness, and depression, and sometimes pain in the back and neck, and convulsions. These cases are often fatal.

Probably the meat is infected from being improperly kept: the proteus bacilli multiply in the intestine, and produce toxins, which, however, are destroyed by heat. The bacilli are not usually found in the blood or organs. Similar symptoms are sometimes caused by eating diseased fish, crabs, mussels, tinned foods, cheese, ice-creams, and even potatoes: and the cause has often been found to be one of the above-named organisms.

Treatment.—The stomach should be washed out to prevent further infection: and except in cases of great collapse a laxative should be given to clear out the intestines. Stimulants are often required, such as brandy, ether, and ammonia: and if the diarrhœa is a prominent symptom opium in small doses, 5 or 10 minims of the tincture. In cases of great collapse, normal saline solution should be injected into the subcutaneous tissue.

It should be here mentioned that some foods are poisonous in consequence of organisms which do not cause gastro-enteritis. Thus in some sausages, ham, canned fish and lobsters, oysters and cheese, the *Bacillus botulinus* has been found. The symptoms it produces (*botulism, allantiasis*) concern the nervous system, and consist of paralysis of accommodation, diplopia, ptosis, dysphagia aphonia, and diminished salivary secretion.

SPRUE

Sprue, or psilosis, is a form of chronic catarrhal enteritis which occurs in the tropics, especially in India, China, the Malay States, and other parts of South-East Asia. The patient has soreness of the mouth, flatulent distension of the abdomen, and looseness of

the bowels. The tongue, palate, lips, and buccal mucous membrane are much reddened, with superficial erosions, patches of congestion, and minute vesicles or aphthæ. Saliva dribbles from the mouth, and eating and swallowing are painful. The stools are copious, pultaceous, frothy and fermenting, white or gray, acid, with a disagreeable smell, different from the normal fæcal odour, and containing numerous bacteria. Temporary attacks of acute diarrhœa with watery stools also occur. Marked wasting and anæmia are the result, and unless the patient is suitably treated, death is likely to take place. In fatal cases the stomach and intestines, especially the lower ileum and colon, have been found much thinned and atrophied, with different degrees of erosion and ulceration of the mucous membrane, and degeneration of villi and glands and follicles. The pancreas has been found to have fatty and granular degeneration of its cells, with increase of connective tissue: and in some cases, besides the general wasting of the tissues, the muscles have shown pronounced atrophy.

Treatment.—The majority of cases can be successfully treated by an absolute milk diet, amounting to three, four, or five pints daily: and this should be continued for many weeks after the last symptom; otherwise a relapse may take place. Strawberries may also be given freely in most cases. For the distressing condition of the mouth and tongue, borax preparations such as the glycerinum boracis, and a lotion containing 5 per cent. of cocain, are useful.

DIPHTHERITIC OR PELLICULAR ENTERITIS

The mucous membrane of the intestine is generally much injected, and presents numerous white patches of membrane. These lie usually along the edges of the valvulæ conniventes, leaving free the depressions between the valvulæ; but they may be more extensive. This form is seen sometimes as a complication in other diseases—*e.g.*, pneumonia, typhoid fever, cirrhosis of the liver, purpura rheumatica, and Bright's disease. The symptoms may be masked by those of the general disease, or may be similar to those of catarrhal enteritis.

PHLEGMONOUS ENTERITIS

In this form all the coats of the bowel are involved, including the serous coat or peritoneum. There is generally intense redness and vascularity, the mucous and submucous coats are thickened, softer, and more friable than natural, and the peritoneum is vascular, sticky, or covered with lymph. It may arise from mechanical interference with the bowel or its circulation by intussusception or strangulated hernia, or from the spread of inflammation from adjacent parts. In intestinal obstruction, also, the bowel above becomes distended from retained fæces, and ultimately its walls inflame, numerous ulcers may form, known as *distension ulcers*, and frequently the bowel ruptures. Cases are,

however, recorded of localised enteritis involving all the coats of the bowel, in which none of these mechanical causes were discoverable. For several inches the coats of the bowel have been much swollen, infiltrated with pus or lymph, and containing minute hæmorrhages. These cases were no doubt due to infection with pyogenic organisms.

Symptoms.—These are often the result of the accompanying peritonitis, and consist of pain, vomiting, local tenderness, collapse, distension of the abdomen, and febrile reaction. The purely infective cases above mentioned have presented the symptoms of intestinal obstruction.

Treatment must be directed to the primary cause, and in its absence is practically the same as that of peritonitis.

ULCERATION OF THE SMALL INTESTINE

Under this heading it is only necessary to refer to the various conditions in which ulceration may take place. The different sections of the small intestine are very differently liable to this particular process. The duodenal ulcer, with its close relations in causation to gastric ulcer, has been mentioned. In the lower part of the ileum occur the ulcers of typhoid fever, of tubercular infection, and rarely of dysentery. Catarrhal enteritis also produces either small follicular ulcers or occasionally larger lesions. The jejunum is of all parts of the bowel the least liable to ulcer, but even there ulcers have occurred, when, after the operation of gastro-jejunostomy, the jejunum is, as it were, turned into a duodenum, and receives straight from the stomach the acid chyme unneutralised by pancreatic secretions. Such ulcers may cause perforation, and so lead to fatal peritonitis unless promptly treated.

COLITIS

Inflammation of the colon presents the same varieties as are seen in other mucous membranes, and may thus be catarrhal or ulcerative. Catarrhal colitis is often a part of a general entero-colitis, arises from the same causes, and has very similar symptoms —namely, pain, distension, tenderness, and frequent motions in which mucus, and even occasionally blood, are present. If the lesion is near the rectum, there may also be tenesmus. Catarrhal colitis may exist in an acute or chronic form; and the treatment is not materially different from that of enteritis.

MEMBRANOUS COLITIS

This is also known as *mucous colitis*, *muco-membranous colitis*, and *mucous colic*, and is characterised by the discharge *per rectum* of large pieces of membrane or *casts*. It occurs most frequently in

middle-aged females, but is also not very infrequent in children. There is commonly troublesome constipation with abdominal discomfort, and other symptoms of chronic dyspepsia; and the discharge of membrane is preceded by severe griping pains.

The casts may be several inches or even feet in length, and an eighth to a quarter of an inch in thickness; they are yellowish-brown, transparent, and gelatinous. They consist of mucus, in which are embedded cylindrical epithelium, some round cells, and crystals of cholesterin and ammonio-magnesian phosphate. The lesion is regarded as a catarrhal colitis, with atony of the bowel; and by the sigmoidoscope in some of these cases, injection, œdema, and ulceration of the mucous membrane has been seen (Mummery). Cancer of the bowel has been associated with these symptoms sometimes. Apart from these last, recovery occurs in many cases.

Treatment.—Careful dieting so as to supply a food free from all mechanically irritating particles, fibres, etc., slow eating and careful mastication, the occasional use of saline laxatives, or castor oil, and irrigation of the bowel with from one to three pints of warm water, are useful means of treatment. But the disease often lasts for years. It has been relieved in some severe cases by opening the colon in the right loin, and by lavage through the appendix brought to the surface by operation.

ULCERATIVE COLITIS

Both typhoid and tubercular ulcers occur in the cæcum and ascending colon in association with similar lesions in the ileum; syphilitic ulcers occur in the rectum; and the whole length of the large intestine is involved in ulceration with different forms of tropical dysentery.

But cases of *ulcerative colitis* occur outside the tropics which cannot be traced to any of the above infections, and which are mostly sporadic. They are seen in both sexes, and at all ages, though much more common in adults. Often there is no antecedent condition of ill-health, but some cases have occurred in connection with a chronic septic disorder, and in others colitis has been the final event of a chronic nephritis. The bacteriology of this disease is not with certainty known: but it is clear that the usual dysenteric organisms, Amœba coli and B. dysenteriæ, are not generally present. The bacillus coli, other pyogenic organisms, and the toxins of antecedent septic conditions, have been suggested as causes. The epidemic occurrence of these or allied cases has been noticed in public institutions, under the name of *asylum dysentery;* but in them the resemblance to tropical dysentery is perhaps closer, and the bacteriological evidence is rather in favour of their identity.

Symptoms.—These come on gradually or more acutely, and consist of paroxysmal griping pain and diarrhœa, with slimy, offensive, fluid motions, containing blood not mixed with the fæces, but no great quantity of mucus. They are often dark, sometimes light

so as to resemble typhoid motions, and rarely consist of bloody mucus alone. Sometimes shreds or sloughs are present. Tenesmus is present in a minority of the cases only: the abdomen is often distended. The patients become sallow, have irregular pyrexia, frequently vomit, lose strength and flesh, and very often die, either from perforation or exhaustion.

The duration is from a few weeks to several months.

Morbid Anatomy.—The colon is the seat of large, irregular, confluent ulcers, with intervening vascular, swollen or pigmented mucous membrane, here and there undermined by the ulcers. Sloughs may be found adhering to the surface; and occasional smooth patches indicative of scarring are found. The general resemblance to dysentery is very close. Hepatic abscesses occur, but they are rare.

Diagnosis.—Clinically the cases often resemble typhoid fever, in the fever, the swollen abdomen, and loose evacuations, but the Widal reaction, the spots, and the typical course of temperature are absent. From tropical dysentery they seem to differ in the relatively small amount of mucus in the stools, in the less frequency of tenesmus, and, from bacillary dysentery, in the absence of serum reaction against Shiga's bacillus. In children intussusception has been simulated. In any case, the ulceration of the rectum and lower colon may be demonstrated by the finger and the sigmoidoscope.

Treatment.—In mild cases the treatment should be that of diarrhœa—rest, bland liquid nourishment, astringents, bismuth, antiseptics, and opium. These are, however, rarely sufficient, and one has recourse then to rectal injections of silver nitrate, argyrol, bismuth carbonate, or boric acid. If this does not suffice, and it may fail because the injections do not reach sufficiently high, an opening may be made at one or other part of the large intestine, in order to irrigate more thoroughly with saline, or weak solution of boric acid. The openings in the cæcum and appendix (cæcostomy, appendicistomy) probably give the best results. More complete operations to exclude the colon will probably fail on account of the diseased condition of the bowel.

APPENDICITIS

(*Perityphlitis*)

Inflammation of the appendix cæci is due to infection by microorganisms probably set up by local injury or disturbance of its vascular structures.

Ætiology.—It is much more frequent in early life than in middle or old age; and in the male than in the female sex. It often appears to be determined by indigestion, or the use of indigestible foods, and occasionally by cold, or by injury. But in numerous instances no cause can be assigned. The greater prevalence of the complaint in recent years, though generally admitted, is quite unexplained.

Pathology.—The appendix normally contains, like the bowel, numerous micro-organisms, of which the *bacillus coli communis* is the most important. As a rule, however, they are not virulent, or they are prevented by the normal movements and secretions of the bowel from exerting any influence upon the tissues. If, however, any local injury is done to the appendix, or if the lumen is obstructed by a foreign body, or by kinking of the structure, the cavity is distended, the vascular and lymphatic system is deranged, and bacterial invasion becomes possible. Thus appendicitis may have as immediate determinants, besides the presence of organisms: (1) The spread of catarrh from the cæcum to the orifice of the appendix, obstructing this so that the cavity is distended by retained secretions. (2) Foreign bodies in the cavity of the appendix, irritating it, or obstructing the lumen. This may be a cherry-stone, orange-pip, seed, bristle, or similar substance. In many cases a concretion is found of the size of a pea, or plum-stone, yellow or gray in colour, and consisting of fæcal matter, mixed with mucus, lime-salts, and numerous bacteria. This is now regarded as being formed subsequently to the catarrh of the appendix. (3) Torsion of the appendix and strangulation of its vessels by undue distension of the cæcum. Besides the bacillus coli communis, streptococci, staphylococci, bacillus pyocyaneus, and other pyogenic organisms, the tubercle bacillus, the typhoid bacillus, and actinomyces are sometimes concerned in the production of appendicitis.

The results are very various. In the appendix itself may be noted infiltration, thickening of its coats, distension of its cavity with catarrhal products or pus, suppuration of its substance, and finally ulceration and gangrene. Thus an inch or more of the extremity of the appendix may be found of yellow, gray, or greenish colour and bloodless; or at one or two points in the length of the appendix a small slough may be formed. In most of these conditions the trouble spreads almost necessarily to the peritoneal covering, and to the sub-peritoneal connective tissue. The peritonitis thus arising is mostly localised and adhesive, matting the appendix to the adjacent bowel, and forming thus more or less definite resistant masses in the right iliac region. If the connective tissue is involved it will be also infiltrated, and assist in the production of the swelling. The inflammatory products may be absorbed, or may suppurate: in the latter case, the abscess, if untouched by the surgeon, may burst externally through the skin, or open into the cæcum, bladder, vagina, rectum, or peritoneal cavity, its course being probably determined to some extent by the anatomical position of the appendix in the subject of the disease.

The appendix sometimes perforates or sloughs before adhesive peritonitis has occurred, and then a general peritonitis, of a very fatal kind, is rapidly determined. Occasionally, a peritonitis, at first local, gradually extends with the formation of peritoneal abscesses in different parts of the abdomen. Infection may thus

spread along the ascending colon to the upper surface of the liver, forming a right subphrenic abscess; or to the lower surface of the liver, and the foramen of Winslow: or if the appendix is on the left side of the cæcum, the infection may reach the descending colon, and spread along it to the spleen and left subphrenic region. Sometimes a pelvic abscess is found, and the neighbouring organs are involved in inflammation, such as the bladder and the pelvis of the kidney. Exceptionally, infective organisms are carried to the liver by the portal vein, and suppurative pylephlebitis and hepatic abscesses are the result.

If sloughing or suppuration do not take place, the apparent subsidence of the inflammation is by no means always the end of the disease: and in many cases from six months to two or three years after the attack, acute inflammation is again lighted up, and either terminates in one of the above processes, or subsides again, perhaps to be active after yet another interval.

Symptoms.—The onset is often somewhat sudden. Spontaneously, or after some such indiscretion as has been referred to, the patient is taken with severe abdominal pain, at first diffused over the abdomen, but soon more pronounced in the right iliac fossa, with malaise, nausea, and some febrile reaction. The tongue is furred, the appetite fails, there is thirst, and the bowels are constipated; there may be also vomiting. The abdomen may be somewhat distended, but is generally rigid; and there is tenderness in the right iliac fossa. This tenderness is often definitely situate at a point about three inches from the right anterior superior spinous process, on a line drawn from this process to the umbilicus—*McBurney's point;* and in this neighbourhood, after a time, a certain amount of resistance can be felt. These symptoms may continue for a few days, the vomiting, pain, and tension may diminish under treatment, and the trouble may subside.

In some cases, however, beginning like this, and in others of which the onset is more gradual, the localised resistance becomes a definite tumour, bounded externally and below by the crest of the ilium and Poupart's ligament, and extending by a convex border half or two-thirds of the distance from Poupart's ligament to the umbilicus. It is often quite dull to percussion, and sometimes it has a modified tympanitic note; while the rest of the abdomen is supple and resonant. The temperature may rise to 103° or 104°, the pulse to 100 or 120, but the patient is generally free from any septic or typhoid condition. The pain may be irregular or paroxysmal, and often shoots down the right leg. In its further progress the tumour may subside, gradually becoming less definite and smaller, so that it disappears in from ten to twenty days from the time it was discovered, while the fever and other unfavourable signs diminish. On the other hand, the swelling may suppurate, with still further enlargement, increasing pain, discomfort, and illness, oscillating temperature with or without rigors, profuse sweating, and all the other indications of septic absorption.

Fluctuation is then generally felt, and occasionally the tumour becomes resonant on percussion, from decomposition and formation of gas in the interior. Spontaneous recovery may even now take place by rupture of the sac into the alimentary canal, when the pain and discomfort are quickly relieved, and a quantity of pus may be noticed to pass by the rectum. Generally, however, the abscess has to be opened by the surgeon; and the convalescence may be slow, as often the sinus is a deep one, and there is much surrounding infiltration. Rupture may take place into the peritoneum, into the bowel, vagina, or other cavity. In the first case the symptoms of general peritonitis will succeed; in the others the swelling in the right iliac fossa will subside, pus is discharged with the stools, or *per vulvam*, and the patient gradually recovers.

In other cases beginning like the first, neither is there evidence of a general peritonitis, nor the well-defined mass as above described; but there is fulness or resistance in some part of the right half of the abdomen, local or general pain and distension are present, and vomiting and septic conditions persist. In such a case there is probably a deep-seated abscess, which may indeed be felt *per rectum* or *per vaginam*, or by its proximity to the bladder may cause frequent micturition.

Some variations occur in local conditions.

In cases of early *sloughing* of the appendix, the local indications may be entirely absent, or so slight that they are scarcely noticed by the patient, or of such short duration that the case is from the first, or quite early, one of general peritonitis (*see* Peritonitis).

The pain at the onset is not always strictly over the appendical region: it may be on the left side, it is frequently umbilical, and it may begin on the left side, and go to the right side. Tenderness and hyperæsthesia of the skin, though frequently over the cæcum, may also be found at other parts of the abdomen. Extension to the general peritoneal surface is usually indicated by increased extent of tenderness, and by immobility of the whole abdomen on respiration. The general condition of the patient also varies a good deal in different cases. The pulse is generally quickened and the temperature is raised: but sometimes the temperature is low, though the pulse is rapid, and this is generally regarded as a sign of severe infection. And, generally, no doubt the pulse-rate is a more important indication of severity than the degree of temperature.

In the relapses of appendicitis, which have been already mentioned, the symptoms are precisely the same as occur in primary attacks; but the liability to general peritonitis is probably less because adhesions will have formed around the lesion. In the intervals, however, deep pressure in the right iliac fossa will sometimes cause a little pain, and some thickening may even be felt.

Diagnosis.—An apparently spontaneous acute general peritonitis in a boy or girl is nearly always the result of appendicitis: in older patients many lesions may be confounded with it. Nearly all the

causes of acute abdominal pain and collapse have been at different times mistaken for it, such as perforation of gastric and duodenal ulcers, acute hæmorrhagic pancreatitis, pyosalpinx, cholecystitis, and renal calculus. The past history, the seat of maximum pain and tenderness, and the local conditions as ascertained by examination externally and *per rectum*, must be carefully considered. Among less severe conditions, neuralgia of the lower abdominal nerves, and the gastric crises of tabes dorsalis, may resemble it; and both in children and adults acute pleurisy and pneumonia at the base of the right chest may cause pain sufficiently low down in the right flank to lead to a diagnosis of appendicitis. Typhoid fever has sometimes been mistaken for appendicitis; and less often appendicitis for typhoid: the latter is the more dangerous error. As a rule, in typhoid there is no acute iliac pain nor vomiting, but sometimes these signs have been present and the difficulty has arisen. The question arises too early for the application of the Widal test, but rose-spots or an active diarrhœa may give the right clue. A leucocytosis in excess of the normal, especially of the polymorphonuclear variety, would be strongly in favour of appendicitis. It will be remembered that an appendicitis may have its origin in typhoid ulceration of the appendix itself.

At a later stage, when a tumour has formed, this has to be distinguished from fæcal accumulations, malignant growth of the cæcum, movable kidney, inflammation of the pelvic organs in women, and psoas abscess. If there is dulness of percussion note over the appendical abscess, it is continuous with the groin or Poupart's ligament, and no line of resonance can be found between them. The leg is not drawn up as it is in psoas abscess.

Prognosis.—In a great number of cases, appendicitis subsides entirely under simple treatment. Of these, however, a certain number relapse, and the second or third attack may be very much more serious than the first. If from the occurrence of a swelling in the right iliac fossa, with a generally supple abdomen, it may be inferred that the mischief is localised, the prognosis is more favourable. This itself may subside, or may be treated surgically. The prognosis is most unfavourable when general peritonitis has already occurred, or when, with few and obscure local signs, the patient has a rapid pulse, serious prostration, and other signs indicative of general toxæmia.

Treatment.—The facts that appendicitis may progress to the stage of gangrene with so few symptoms, and that in any case extension to the general peritoneum may take place with extreme rapidity, make it desirable to consider from the first whether the appendix should be removed at once or not. When the onset is very acute, with much pain and tenderness, vomiting, quick pulse and prostration, this is probably the wisest course. If the symptoms are less urgent, it may be justifiable to wait: and the patient should be put to bed, and fed only on milk, Benger's food, and similar articles of diet: hot boric lint or fomentations should be

applied, and all aperients should be withheld, except a simple enema if the bowels have not recently acted. If a tumour forms in the right iliac fossa, while the rest of the abdomen is supple and painless, the medical treatment may be continued, though a time will probably soon arrive when surgical assistance will be required to evacuate pus. In other cases where no operation has been performed at the onset, and no localised tumour can be recognised, and yet there is reason to believe, from continued pain, from tenderness on deep pressure, fulness or resistance in the right iliac fossa, or in the right flank, distension of the abdomen generally, fulness or resistance in the rectum, or irritation of the bladder, that there is deep-seated suppuration, laparotomy should be performed. And operation is imperative at the first sign of an extension of inflammation to the peritoneum generally.

Whether every case which has recovered under medical treatment should be operated on some months later to prevent recurrence is an open question: but if a second attack occurs, certainly the opportunity should be taken to operate: and if this subsides without operation, what remains of the appendix should be removed some weeks after recovery. Similarly, after a first attack, if recovery is delayed, or incomplete, with recurrent local pain, febrile attacks, tenderness and thickening, the operation should be performed.

TUBERCLE, NEW GROWTHS, AND SYPHILIS OF THE INTESTINE

Tubercle.—Tuberculosis of the intestine has been already referred to under Phthisis, but it may occur independently, especially in children, to whom the infection is often conveyed by milk. In either case the lesions occur chiefly in Peyer's patches in the ileum, and in the solitary follicles of the ileum and colon. The process is the same as elsewhere, cell-proliferation, caseation, necrosis, and ulceration; and the ulcers in the ileum may be of great extent, being round or oval, and frequently running transversely round the gut, rather than along it. The surface is irregular, with thickened edges, and the serous surface corresponding to it generally presents small white tubercles in some number. The symptoms associated with tubercular ulceration of the bowel are pyrexia and diarrhœa. The motions are generally abundant, pultaceous, fatty, and yellow in colour. Sometimes they are more fluid, and still yellow, and if the abdomen is distended there may be a close resemblance to those of typhoid fever. Hæmorrhage and perforation are rare. The treatment has been indicated (*see* p. 540).

Fistula in ano is no doubt sometimes tubercular in origin.

New Growths.—The new growths of the intestine are chiefly *adenoma* and *carcinoma*, and rarely *lymphosarcoma* (*see* p. 721). Carcinoma is the most frequent and important. It is much more common in the large than in the small intestine, and affects

especially the cæcum, the flexures of the colon, and the rectum. It forms rounded or nodular hard tumours, painful and tender, at first mobile but later fixed by adhesions, or by extension to adjacent parts. Ultimately growing into the lumen of the bowel it causes obstruction (*see* p. 743). If the growth is recognised before obstruction occurs, or adhesion has taken place, its removal by operation may be possible; but it may be confounded with other lesions. Affecting the cæcum, it may resemble a chronic appendicitis, or enlarged glands; in the colon an enlarged kidney, an enlarged gall-bladder, or an aneurysm if it overlies the aorta.

Cancer of the rectum, which occurs chiefly in elderly people, is liable to be mistaken for dysentery, the symptoms being local pain, straining or tenesmus, and the passage of small quantities of mucus, sometimes stained with blood. An examination with the finger will enable one at once to recognise the mass of new growth blocking the passage not far from the anus, and yielding the above-mentioned fluid.

Syphilis.—Syphilis rarely affects the alimentary canal between the pharynx and the rectum; but it sometimes causes stricture in this last situation. Gummata form in the sub-mucous tissue, and slowly lead to cicatricial contraction. They occur commonly in women, and little may be known of them till symptoms of stricture are observed, and the constriction is recognised by digital examination.

INTESTINAL OBSTRUCTION

The intestine may be obstructed in several ways: (1) Foreign bodies, large gall-stones, or collections of fæcal matter in its interior; (2) intussusception or invagination; (3) changes in the intestinal walls, such as strictures caused by healed ulcers, or by malignant growths; (4) volvulus; (5) strangulation by bands or through apertures; (6) diminution of the calibre due to traction on the intestine, or to compression from outside in various ways.

Pathology. *Foreign Bodies.*—Among the foreign bodies found obstructing the bowels are fruit-stones, pebbles, coins, bullets, pins, needles, hooks, and false teeth. Sometimes large masses are formed of vegetable fibre, wool, or husks of oats, matted together. It is especially in lunatics that foreign bodies of this kind are found. Occasionally a large gall-stone is the cause of a fatal obstruction; or it passes *per anum* after more or less difficulty. Such gall-stones may be two or three inches in length by three or four in circumference; they are formed in the gall-bladder, and reach the bowel, not through the bile-duct, but by ulceration through the walls of the gall-bladder and the duodenum. They commonly obstruct the small intestine, especially the lower part of the ileum or the duodenum. Fæcal masses may accumulate in the same way as has been described under Constipation, and form a serious obstacle, in the rectum, sigmoid, or colon. They are more common in women

than in men, and are mostly met with in adults. Masses of magnesium salts, after the extreme use of carbonate of magnesium, have been known to form in the bowel and give rise to obstruction.

Intussusception.—This presents special features which make it desirable to consider it separately (*see* p. 750).

Strictures.—These occur both in the small and large intestines; they arise either from contraction of cicatrices of ulcers, or from new growths in the intestinal walls. Of the different forms of ulceration, dysenteric and catarrhal ulcers most frequently give rise to stenosis, and typhoid ulcers rarely, if ever, do so. But in many cases of stricture there is no evidence as to what form of ulceration has preceded it. Simple strictures are generally single. Occasionally, they follow the reduction of a strangulated hernia, or injuries to the abdomen. The new growths causing stricture are, as a rule, of malignant nature, and usually *cylindrical epitheliomata.* In the majority of cases it is a primary growth, but occasionally it is secondary or extends from adjacent parts. The form it commonly assumes is that of a band or ring round the bowel, by which the internal circumference is considerably reduced, even to the size of a cedar pencil, but rarely completely obliterated. The longitudinal extent of the growth is often not more than one or two inches; the inner surface is frequently ulcerated. Simple tumours, such as *adenomata* and *fibromata,* are only occasionally the cause of intestinal obstruction.

Strictures, whether simple or malignant, are more common in the large than in the small intestine; this is especially true of malignant disease. And in the large intestine itself the sigmoid flexure is most often the seat of stricture; the descending colon comes next in frequency, and the hepatic flexure is somewhat more often attacked than the splenic flexure. In nearly 70 per cent. it is the sigmoid or descending colon. Females are somewhat more often affected than males; the patients are generally middle-aged, malignant cases being usually about forty, and cases of simple stricture somewhat younger. Strictures are essentially of slow development, and the gradually increasing obstruction influences the condition of the bowel above. This becomes distended by the accumulation of fæces, and hypertrophied in its efforts to force its contents past the stricture. Fæcal matter naturally accumulates, but is from time to time passed through, sometimes after symptoms of almost fatal intensity; occasionally, however, foreign bodies, such as fruit-stones, of larger diameter than the aperture, may collect above it. Increasing distension and pressure from within may ultimately lead to ulceration, gangrene, and perforation of the gut above the stricture. In strictures of the sigmoid or descending colon, fæcal matter often accumulates in the cæcum.

Volvulus.—By this term is meant the twisting of a loop of bowel upon itself, so that the two portions at the ends of the loop cross and strangulate each other. It is most common in the sigmoid flexure, or pelvic colon, which may form a loop sufficiently free,

from the length of its meso-colon, for the purpose. More rarely the cæcum is twisted on its vertical axis so as to cause obstruction, or is bent up in front of itself; and occasionally the small intestine forms a volvulus of the same kind as that described in the sigmoid.

In another form of volvulus two loops help to strangulate one another; so, for instance, the sigmoid flexure may be doubled round a loop of the ileum, or two loops of the ileum round one another. Volvulus occurs in males more often than in females, and mostly between the ages of forty and sixty.

Strangulation by Bands and through Apertures.—This class of cases is precisely analogous to ordinary cases of hernia, and they are often described as internal strangulation and internal hernia; a loop of intestine, commonly the ileum, slips through an aperture, and is strangulated by the margin of the aperture grasping its neck. Some apertures are slits in the omentum or mesentery. Another kind of aperture is the opening into a fossa, or pouch of peritoneum, such as are found in connection with the duodenum, and the ileo-cæcal junction: and a hernia may occur into the lesser cavity of the peritoneum through the foramen of Winslow. But much more frequently the constricting ring is formed by a band of adhesion stretching from one part of the abdomen to another, under which the loop of gut passes, or by the same band forming more or less complicated loops in which the gut is involved. Bands of this kind arise from a former peritonitis, probably local in extent, and often in connection with the pelvic viscera or the appendix cæci. They are often solitary, though they may be accompanied by other adhesions not forming bands. The pedicle of an ovarian tumour may strangulate the bowel.

A frequent cause of this form of obstruction is the congenital abnormality known as *Meckel's diverticulum.* This forms a finger-like projection from the unattached side of the ileum, from two to four inches in length, and half to three-quarters of an inch in diameter. It has the same serous, muscular, and mucous coats as the ileum, and is a remnant of the omphalo-mesenteric duct, by which the primitive alimentary canal communicates with the yelk-sac. It arises from the ileum, at a point eighteen to twenty-four inches from the cæcum, and its blind termination is generally free; it may be attached by a fibrous band to the anterior abdominal wall at the umbilicus, or to the mesentery, or to the peritoneal surface at some other point. A ring is thus formed, through which a loop of gut may slip, and then become strangulated.

When once the loop has slipped through, strangulation is favoured by everything which increases the contents of the loop, such as more air or intestinal liquid; and not infrequently the loop itself becomes twisted like a volvulus.

Strangulation by bands and apertures is more frequent in males than in females, and occurs at all ages, but mostly between the ages of twenty and forty. Among cases occurring in early life, strangulation by Meckel's diverticulum is the most common.

Compression and Traction.—This class includes the following forms of interference with the calibre of the gut: Acute kinking due to traction upon an isolated band, or an adherent diverticulum; adhesions retaining the bowel in a bent position; adhesions compressing the gut; matting together of several coils; changes effected in the intestinal coils due to simple traction; and narrowing of the bowel from shrinking of the mesentery after inflammation. The cases are comparatively rare; they concern the large intestine and small intestine with about equal frequency; and they are likely to be preceded by a history of peritonitis.

Effects of Obstruction upon the Bowel.—In a fatal case of acute obstruction of the intestine, the bowel above the seat of obstruction is found enormously distended, while that below is collapsed and empty. The distension begins immediately above the constriction, and affects the bowel for a greater or less distance, according to the severity or duration of the obstruction. Thus, in obstruction at the sigmoid, the whole colon and much of the small intestine are affected; in obstruction of the ileum, the small intestine is distended and the colon is collapsed. In the upper distended portion is a quantity of fæcal matter, light brown or yellowish-brown in colour, and of uniform thick liquid consistence; and this is the same whether the obstruction is in the small or large intestine; there is never sufficient absorption by the intestinal vessels to form the harder and drier fæces of health. In chronic cases the distended bowel becomes gradually hypertrophied from its efforts to overcome the obstruction. If this is unrelieved, ulceration, sloughing, and rupture or perforation take place, with peritonitis as a result. In acute strangulation, sloughing may occur at the seat of constriction, from the direct interference with the circulation; in the chronic obstruction of strictures the bowel yields in the distended portion above. Where large fæcal accumulations are the cause of obstruction, the scybalous masses irritate the mucous membrane and set up catarrh and ulceration, forming so-called *stercoral ulcers*.

General Symptoms of Obstruction.—The symptoms of intestinal obstruction are vomiting, constipation, pain, and distension of the abdomen. The special feature of the *vomiting* is its *stercoraceous* or fæcal character. At first the contents of the stomach are discharged, and subsequently bilious matter; but comparatively early in acute cases, and with the final obstruction in chronic cases, the vomited matter consists of light or dark brown thick liquid, with a distinct or even strong fæcal odour. This rejection of the contents of the bowel has been ascribed to its increased peristaltic movements driving downwards the liquid which is next to the intestinal wall, while a central current is established in a reverse direction—that is, towards the stomach.

The *pain* of obstruction is variable. In acute cases it is very severe, generally paroxysmal at first, and not becoming continuous until the obstruction is complete. Its situation is sometimes

determined by the position of the lesion in the abdomen, but often it is referred to the umbilical region, though the strangulation may be in quite another part of the abdomen. The pain in chronic obstruction may be very slight, but it is aggravated when obstruction increases to a marked degree. Tenderness is not generally present until peritonitis sets in.

Constipation is an important feature in obstruction, though not in itself conclusive, as it is present in other conditions. It is generally absolute from the time of obstruction—not only is there no motion, but also no flatus whatever. Occasionally, however, the lower bowel may contain fæces at the time the obstruction occurs, and these may be discharged, or removed by an enema.

But a more complete description must be given of the symptoms and course of intestinal obstruction; and we must distinguish between acute cases, of which the strangulation by a band is the most typical example, and chronic cases, of which malignant stricture of the sigmoid is the best instance.

Symptoms of Acute Obstruction.—In a case of strangulation by a band, the patient is seized with intense pain in the abdomen, generally in the neighbourhood of the umbilicus; he may be walking about, or having a meal, or he may be awakened from sleep. Sometimes the attack is attributed to a strain, or to some unaccustomed or indigestible food taken some hours previously; but it is often impossible to prove the connection. The patient then vomits, either directly or within a short time, the vomited matter being the contents of the stomach. The pain is almost continuous, and vomiting is excited by every attempt to take food. The abdomen generally becomes tense, but the actual distension varies with the position of the obstruction; if this is in the upper part of the small intestine, the abdomen may be flat, or distended only at the upper part, above the umbilicus; if the lower part of the ileum is strangulated, the abdomen is uniformly enlarged. Neither motion nor flatus is passed *per anum;* and the vomiting, at first gastric, then bilious, becomes ultimately stercoraceous.

The effect upon the patient is very grave. Collapse soon sets in; the face is drawn, the eyes are dark and sunken, the pulse small and quick, the temperature normal or subnormal, and much flesh may be lost in a few days. The tongue is dry, and there is constant thirst. The urine is scanty and high-coloured; its quantity tends to be less the higher the seat of the obstruction—a fact which is to be attributed to the generally more constant vomiting, so that but little food or fluid is absorbed into the system. If the condition is unrelieved death supervenes, either from exhaustion, or from peritonitis, of which a general diffused tenderness may be the chief indication. The duration of the case is from four to six days.

The forms of intestinal obstruction which commonly cause acute obstruction, besides strangulation by bands and apertures, are intussusception, volvulus, impaction by gall-stone, and some forms of acute kinking by adhesions. The attempt to distinguish between

these cases is often unsuccessful; as the results of an obstruction must depend much more upon its position in the length of the intestine, upon the rapidity of its occurrence, and upon its completeness than upon the actual tissue changes. And the practical value of the differential diagnosis is not great, as there are few cases, if any, which can be safely left without an operation. Acute kinking produces symptoms like those above described, but the pain is generally less continuous, the case is less rapid, and the symptoms more variable in intensity. In complete obstruction by gall-stones, the onset is usually sudden, and the pain is severe and continuous with exacerbations. Vomiting appears early, is abundant, and becomes stercoraceous. There may have been previous illnesses attributed to gall-stones. Other foreign bodies less often cause acute obstruction. In volvulus there is very great distension of the abdomen and embarrassment of respiration; but pain, vomiting, and collapse are often not so severe as in other cases, and the duration may be from four to ten or fifteen days. Intussusception has its special features, described later.

Symptoms of Chronic Obstruction.—In chronic obstruction such as is due to malignant disease of the sigmoid flexure or of the descending colon, the symptoms are at first only indicative of a moderate interference with the passage of fæces; there are some local pain, and occasional vomiting, not particularly related to the ingestion of food. Constipation occurs irregularly, but it can be overcome by aperients. From time to time the constipation is very troublesome, vomiting is more frequent, yet not stercoraceous, the abdomen becomes greatly distended, and the hypertrophied coils become visible in peristaltic movements on the surface of the abdomen. When the distension mainly affects the colon, as in the case of sigmoid cancer, the transverse colon bends downwards in the middle and forms two enormous vertical coils. When the small intestine is chiefly distended, and the colon is collapsed, the distended coils often lie transversely across the abdomen. With the peristaltic movement can be heard gurgling sounds, or borborygmi.

After a week or ten days of such symptoms, some fluid motions may pass, and then quickly several large evacuations of liquid fæces, by which the abdomen is rapidly reduced to its normal capacity, and all the symptoms are relieved. This sequence of events may recur more than once, but in some such attack the constipation becomes complete, nothing is passed *per anum*, vomiting is more frequent and becomes stercoraceous, the abdomen is enormously distended, with visible moving coils, there is severe pain of griping character, and, after some days, it may be as many as ten or twelve, death takes place from exhaustion, or from rupture of the bowel and peritonitis. If the case is seen early, a tumour can be sometimes detected in the left iliac fossa, or one or other flank, but its recognition may be quite impossible when the abdomen has become much distended. The patient may also present the loss of colour and emaciation so common in cases of malignant disease.

The chronic cases which produce somewhat similar symptoms are other forms of stricture of the large intestine, strictures and growths of the small intestine, most forms of compression, traction, and matting of the gut by adhesion, some cases of volvulus, compression of the gut by tumours outside it, some cases of impaction of a foreign body, and fæcal accumulations.

Sufferers from *fæcal accumulations* have generally had previous attacks of constipation, which have only been relieved by strong aperients; and at length even these are useless. The patient then has indigestion and flatulence; the abdomen swells, it may be to an enormous extent, and causes dyspnœa by its pressure on the diaphragm, while the mass of fæces may press upon the lumbar or sacral plexus, or the abdominal or pelvic veins. Sometimes the fæces excite catarrh of the bowel, and a little thin fluid escapes, which may be mistaken for a genuine relief. Nausea, eructations, and vomiting follow, and the coils may be so distended as to be visible on the surface. Occasionally the vomiting becomes stercoraceous, and death takes place from exhaustion. In many cases a tumour due to the accumulated fæces can be felt: this is especially the case if the obstruction is in the large intestine, when the mass often occupies the cæcum and the ascending colon. The tumour is uneven, rounded or elongated, and generally painless: often it can be indented by the finger, or is doughy in consistence. The duration of these cases may be several months.

Position of the Stricture.—The differences to be noted between strictures of the small intestine and those of the large are, that in the former vomiting occurs earlier, and is more determined by the ingestion of food: in the latter distension is greater, and the proximity of the stricture or growth to the anus may lead to alterations in the shape of the motions, which may be ribbon-shaped; and tenesmus is frequently present. The stools, moreover, often contain blood. If the abdomen is distended, the prominence is greatest in the middle line in obstruction of the small intestine or of the ascending colon; but more general if the sigmoid or the descending colon is the seat of disease.

Diagnosis of Obstruction.—Acute abdominal pain and distension with vomiting and constipation are common results of so many abdominal lesions, that diagnosis is often very difficult. Those which may be wrongly taken for intestinal obstruction are especially acute peritonitis from perforation or appendicitis, and acute hæmorrhagic pancreatitis. In the former case the following may serve as guides: In peritonitis there is diffused tenderness and general distension; the temperature is often elevated; the vomiting is perhaps less severe than in strangulation, and rarely fæcal. The onset may have been preceded by symptoms pointing to the appendix. Fæcal vomiting forms the strongest evidence in favour of obstruction, but the vomited matter must, if possible, be seen, as friends will often represent as fæcal what is merely gastric or bilious. The rectum should be examined by the finger: it is often empty and dilated, or "ballooned" in cases of obstruction.

The gastric crises of tabes dorsalis may resemble obstruction, but the abdomen is retracted and the vomit is not fæcal. A history of similar attacks in the same patient, and the absence of knee-jerks, and of the pupil light-reflex, would speak for tabes. On the other hand, intestinal obstruction itself has sometimes been mistaken for other diseases—*cholera, lead colic, hepatic colic, renal colic, arsenical poisoning,* and even *meningitis.* The possibility of obstruction from an extra-abdominal hernia, whether inguinal, femoral, or obturator, should not be forgotten. The corresponding openings should be investigated: but if, from their freedom, the obstruction can be located within the abdomen, the differential diagnosis as to the exact position is of small importance, as a laparotomy becomes absolutely essential.

Treatment.—When the diagnosis of acute intestinal obstruction is established the operation of *laparotomy* or opening the abdomen should be performed without delay; and the cause should be ascertained and an attempt made to remove it, as, for instance, by reducing a strangulated coil, dividing a band, unfolding or resecting a volvulus, or extracting a gall-stone. Even if the diagnosis between obstruction and peritonitis cannot be determined, the operation is still desirable, as assisting both diagnosis and treatment. If the obstruction cannot be found, or the parts are matted together so that it cannot be dealt with, the bowel must be opened above the obstruction either in the wound employed, or through a fresh opening in the loin, as seems most expedient. If the patient is already so exhausted when the lesion is first recognised, as to make it highly probable that he will sink under a peritoneal operation, the bowels should be simply opened at the most distended part, and a fæcal fistula established. In suspected cases previous to operation, and in cases where for any reason an operation is not performed, the patient should be fed by nutrient enemata; and purgatives must not be given, for they can only aggravate the case by exciting the peristalsis of the intestines to fruitless efforts, whereby the congestion and strangulation of the bowel are actually increased, and the pain and vomiting are rendered more severe. It is rarely wise to give opium or morphia for the relief of pain: it has the serious disadvantage that, while easing the pain and checking sickness, it removes two important symptoms, and may lull to a false security while the fatal mischief is progressing. Locally, relief may be furthered by the application of turpentine stupes; or of flannels wrung out of hot water, and sprinkled with tincture of belladonna, or opium; or of hot linseed-meal poultices.

In *chronic* obstruction, which is chiefly the result of strictures and growths, whether in the small or large intestine, the diet must be carefully selected, with the object of ensuring regular digestion and the easy passage of the intestinal contents through the constriction. Enemata, and occasionally laxatives, may be used to maintain a periodical evacuation. If an obstinate constipation ensues, and especially if great distension and sickness occur, the

treatment must be assimilated to that of an acute obstruction. Opium may be given, with or without belladonna, while food must be given in only small quantities, or *per rectum*, when relief may be shortly obtained. Eventually, if life is to be prolonged, an operation will become necessary.

For stricture of the colon a colostomy should be done in the right or left loin, according to the position of the obstruction; in some cases the removal of the diseased portion of bowel (*colectomy*) may be desirable. For the small intestine, laparotomy will be probably required, and the bowel must be dealt with by intestinal anastomosis or excision.

For fæcal accumulations, large and frequently repeated enemata generally suffice, but the case requires to be long under treatment by careful diet, exercise, electricity or massage, to restore the bowel to its former power.

INTUSSUSCEPTION

If one segment, say a few inches, of the intestine slips into the portion immediately adjacent, it forms an *intussusception* or *invagination*. It will be at once seen that this must present from without inwards to the centre of the bowel three layers of bowel-wall, of which the innermost may be called the *entering* layer; the outermost, the *receiving* layer or *sheath;* and the portion joining these two, the *middle* layer. The process of intussusception may continue, so that more and more bowel is involved, and this usually takes place by the entering and middle layer moving in uniformly together, and, as it were, dragging in the outer layer after them. In this way, as more of the entering layer disappears into the intussusception, the middle layer increases at the expense of the outer layer. The inner bend, between the entering and middle layers, remains always the same, the most advanced portion of the intussusception; the outer bend, between the middle and outer layers, is constantly shifting. It is clear that any portion of bowel might slip into a segment above, forming an *ascending* intussusception; or into the bowel below, forming a *descending* intussusception. It is with the latter that we practically always have to do.

Intussusceptions occur at any part of the bowel, and have received names accordingly; thus, those of the small intestine are called *enteric*, those of the large intestine *colic* or *rectal*. But at the point of junction with the ileum and the colon two varieties occur—(1) The *ileo-cæcal*, in which the ileum and cæcum pass into the ascending colon, the ileo-cæcal valve forming the most advanced point, the ileum the entering layer, and the cæcum the most advanced part of the middle layer; (2) the *ileo-colic*, in which the lowest part of the ileum is inverted through the ileo-cæcal valve—that is, an enteric intussusception continued into the colon. Of the different forms the ileo-colic is the rarest, and the ileo-cæcal is the most common, forming nearly half of all cases.

Very important changes, dependent on the anatomical relations of the intestines, ensue upon an intussusception. The intussusception, if at all extensive, forms a thick cylindrical swelling, partly from containing three layers of bowel all round instead of one, partly on account of the congestion and œdema to be presently explained. From the mesenteric connections of the bowel this cylinder has a curved shape, since the vessels which supply the inner and middle layers are of the same length as those supplying the receiving layer, and yet have not only to reach the border of the intussusception, but to go into its interior between the inner and middle layers, so that they drag upon the upper end of that part of the bowel. As the intussusception increases it moves farther along the gut, and the internal cylinder of an ileo-cæcal intussusception may even reach the rectum and project from the anus. At the same time the tumour becomes larger. The disposition of the vessels just described leads to their compression and strangulation, and consequently to congestion and œdema of the walls of the intussusception; and even to hæmorrhage from the mucous surface, and the discharge of blood *per rectum*, an occurrence of the greatest value in diagnosis. If the case is not quickly fatal, inflammatory changes ensue in the layers of the bowel, binding them together, and interfering both with the further progress and with the reduction of the intussusception; and, lastly, from the strangulation of the blood-supply to the entering and middle layers, these may become gangrenous, slough off, and be discharged *per rectum*. If this has been preceded by the secure adhesive union of the entering layer to the angle between the outer and middle layers, the canal of the bowel is practically restored, and an actual cure may be the result, though this is very rare; if the union is imperfect, the detachment of the inner cylinder is followed by a fatal extravasation.

Ætiology.—The cause of intussusception is obscure in the majority of cases. Sometimes it has arisen after strains or direct injuries, or after unsuitable ingesta, or in connection with diarrhœa. Intestinal polypi and cancerous tumours have sometimes seemed to favour its occurrence. It may happen at all ages, but is much more frequent in children; and it affects males more often than females in early life, though the difference between the sexes is not so great in adults. Little that is definite can be said as to the immediate mechanism of intussusception, except that it is due to an irregular peristaltic action.

Symptoms.—The onset of an acute intussusception is not unlike that of strangulation by bands—that is, the patient is rather suddenly seized with pain, which is more or less constant, though aggravated from time to time, and griping in character. In the baby, the onset is indicated by screaming. Nausea and vomiting also occur, but constipation is not generally present at first; on the other hand, the bowels are usually moved, and either thin fæces, or (what is especially characteristic of intussusception) *blood* with

or without mucus is passed. Indeed, blood is passed *per rectum* in four-fifths of the acute cases; and often a certain amount of tenesmus is present. The abdomen is not always much swollen, but an examination reveals generally another characteristic feature—the presence of the *tumour* which results from the intussusception. Its position is, of course, related to the site of the lesion; in the more ordinary ileo-cæcal form it is at first situate in the right flank, but as the intussusception increases it is felt in the umbilical region, and is generally oval, cylindrical, or sausage-shaped, lying transversely across the abdomen above the umbilicus. Subsequently it passes into the left flank, and left iliac fossa, and ultimately can be felt by the finger in the rectum, or actually projects from the anus. Sometimes there is complete constipation, much distension, and fæculent vomiting; at others collapse sets in rapidly, and death takes place in twenty-four hours, or from two to five or six days. Death is especially rapid in quite young infants.

But the symptoms are not always so acute; indeed, an intussusception may exist for weeks or even months. In these more chronic cases the extent of bowel involved is generally less, and the canal is not completely obstructed. The bowels may thus be opened, though blood is passed at the same time in about half the cases. The patient suffers from paroxysmal griping pains, not necessarily of great severity. The abdomen is flaccid, and the tumour presents an important characteristic, namely, a varying consistence; so that it hardens simultaneously with the griping pains, but soon becomes soft, and even imperceptible when they subside.

The terminations of the subacute and the chronic cases are various; they may ultimately lead to death by exhaustion, or to complete obstruction with vomiting, constipation, abdominal distension, and visible coils; or they may set up a local peritonitis, followed by the formation of abscess, or by a more general peritonitis; or the intussuscepted portion may separate by sloughing, and so the intestinal canal may be re-established.

Diagnosis.—Spasmodic pain, vomiting, the passage of blood *per rectum*, and the presence of an oval or elongated tumour which varies in consistence from moment to moment, and lies in the course of the colon, or occupies the rectum, are the chief features of intussusception; but the tumour cannot always be felt, especially in infants with a much distended abdomen, unless an anæsthetic is given. *Enteritis* and *dysenteric diarrhœa* in children may resemble it, but there is no tumour, and the blood is less often pure or unmixed with mucus.

Treatment.—An *acute* intussusception should, as soon as possible, be met by an effort at reduction, and though cases have been successfully treated by injection of fluids into the rectum and colon, it is safer and more certain for the surgeon to perform laparotomy, and reduce the intussusception by careful traction. In the longer-standing cases adhesions may render reduction

impossible; and then a portion or the whole of the mass must be resected. If for any reason an operation cannot be performed, the attempt should be made to reduce the intussusception by injection. Chloroform should be given, and a Lund's inflator or a suitable rectum-tube, attached to a small bellows or bicycle pump, is inserted in the rectum, and the buttocks are closed firmly upon the instrument. Air, salt solution, milk, or oil is then pumped in, and its progress along the bowel is watched by the hand placed on the abdomen, when the tumour formed by the intussusception may be felt moving towards the right side of the abdomen, and ultimately disappearing. If liquid is employed it should be warmed to 100° F. and introduced by a soft india-rubber tube and funnel, the latter being raised 3 feet above the anus. Too much force may rupture the coats of the bowel, and for this reason air seems to me preferable to any liquid, since it is less unyielding, and yet can be renewed to any extent required. After the reduction, small doses of opium may be administered, with careful dieting for a few days.

In *chronic* forms the diet must be carefully regulated, and opium may be given to relieve pain, spasm, or sickness. Here, also, inflation may be attempted, but in consequence of secondary changes it may be unsuccessful; and then the severer operation will be required.

ENTEROSPASM

This is a condition of spasmodic contraction of localised portions of the bowel. It affects the colon more often than the small intestine, and mostly either the commencement of the ascending colon or the end of the descending colon. It has been seen, in explorations made on a mistaken diagnosis, that the bowel is contracted for some inches so as to form a hard pale, or bloodless cord. The patients are seized with pain, and suffer from constipation, moderate distension of the abdomen, and vomiting. The pain or discomfort is mostly in the right or left iliac fossa: the abdomen is rigid, and there is tenderness over the affected colon, but no peristalsis is visible. The patients are often neurasthenic, or suffer some mental depression. The attacks last for years.

Diagnosis.—When the pain is in the right iliac fossa, enterospasm may be mistaken for appendicitis; and in other cases for enteralgia, or colic, or if the tumour is recognised, for cancer of the colon. The variation of the tumour, and its subsidence after a time, should prevent this.

Treatment.—In the attacks hot fomentations or poultices should be applied: and belladonna or potassium bromide may be given internally. In the intervals the general health should be treated by tonics, and the bowels should be regulated by mild laxatives.

HIRSCHSPRUNG'S DISEASE

This is a rare disease, of which the feature is chronic dilatation and hypertrophy of the colon. In many cases the symptoms begin in the first few weeks of life, in others in early childhood, and in others later. There is constipation, obstinate and repeated, the bowels remaining unopened for two or three weeks at a time. The abdomen is distended enormously, so that the pressure on the chest may itself be a danger. Through the thinned abdominal walls the distended coils of the colon may be seen, and generally the front of the abdomen is occupied by a vertical loop, with the angle near the ensiform cartilage, which is formed of the pelvic colon. In addition the transverse colon is often distended, and sometimes the ascending or descending colon. Peristaltic movements are visible in the coils. The child is emaciated, and in the older patients defective nutrition and sallow skin are observed. If pronounced in infancy the condition is generally soon fatal, by convulsions or pulmonary disease. Those in whom it is first found at a greater age may live some years, and die of intercurrent disease or toxæmia. After death, the colon is found to be dilated to two or three times its normal diameter, and often much elongated: and in long-standing cases the muscular fibres, especially the circular layer, are much hypertrophied. The part first affected is nearly always the pelvic colon. The actual cause is not at present clear. No persistent organic change is present: but it is believed by some that constantly repeated kinking at the junction of the pelvic colon and the rectum might bring it about. It has been called *idiopathic* and *congenital*, but the justice of either term is somewhat doubtful.

Treatment.—Attempts must be made to keep the colon free. Purgative drugs are of little value: enemata of oil or glycerine may do good if the bowel will respond to them. Strychnine in full doses, electricity and abdominal massage may also help. The diet must be of a kind which will leave little residue, such as milk, fish, eggs, and white bread. If all these fail an operation may be necessary, either a colostomy, or ileo-colostomy, or complete resection of the dilated portion of the colon.

INTESTINAL WORMS

The following are the worms more commonly met with in the human alimentary canal:

Cestoda, or tapeworms (κεστός, a girdle)	*Tænia solium.* *Tænia mediocanellata.* *Bothriocephalus latus.*
Nematoda, or round worms (νῆμα, a thread)	*Ascaris lumbricoides.* *Oxyuris vermicularis.* *Trichocephalus dispar.* *Ankylostoma duodenale.*

Trichina spiralis also develops in the alimentary canal, but its symptoms mainly result from its infesting the voluntary muscles (*see* p. 433).

It is essential to say something of the life-history of each of these worms before dealing with the symptoms which it produces, and the means to be employed for its expulsion.

TÆNIA SOLIUM

Anatomy.—The Tænia solium is a flat, ribbon-shaped worm, very narrow at one end, broader at the other, from ten to twelve feet in length, and divided into a number of small segments. There is no alimentary tube, but two canals extend the whole length of the animal, constituting the so-called water-vascular system. At the narrow end is a globular swelling, or head, not larger than a pin's head, presenting a central prominence, or proboscis, surrounded by a row of twenty-six hooklets; and four suckers are placed at the sides. Below the head is a narrow portion or neck, where the segments are quite small and thin, but they gradually get broader and larger towards the other extremity. As they become larger, these segments acquire sexual characters, and are then called *proglottides:* each one bears male and female organs, the apertures for which are on one edge of each segment, alternately on the one side and the other of the tapeworm. A fully developed Tænia solium may contain about 850 segments, of which only the last 80 to 100 are mature. A mature proglottis measures half an inch in length, by a quarter of an inch in breadth. The uterus is an elongated cavity running the whole length of the segment, and giving off from seven to ten branches on each side, which again branch freely. The ova measure ·03 mm., are slightly oval in shape, and have a thick shell, presenting fine radiating lines visible under the microscope. The embryo develops while the ovum is still in the uterus.

The tapeworm inhabits the small intestine, being attached firmly to the mucous membrane by its head, while the chain of segments lies, partly coiled, along the bowel, as far down as the lower end of the ileum. As the lowest segments become mature they are detached, and are passed with the fæces. The ova may escape during this transit, or subsequently by the decomposition or rupture of the segments, and they thus become scattered on the ground, or on leaves, grass, or elsewhere.

For the further development of the ovum it is essential that it shall be taken into the stomach of an animal; and in the case of the Tænia solium it is the pig that performs this service, swallowing the ova with vegetables, or with the refuse upon which it feeds. Arrived in the stomach of the pig, the shell of the ovum is dissolved by the gastric juice, and the embryo, or *proscolex*, provided with six hooks, escapes to bore its way into the gastric or intestinal vessels, and thus be carried to the liver, muscles, or other part of

the body. In some such situation the embryo remains, and develops into a little globular bladder about the size of a pea, with which is connected, by a narrow segmented neck, a head with hooklets, suckers, and proboscis, precisely like that of the complete tænia. The head and neck, however, are usually retracted or inverted into the centre of the little cyst. These cysts occur in great numbers, in the muscles of the pig, and the flesh so affected is described as "measly pork"; and they are seen occasionally in man, in the connective tissue, in the eye, and elsewhere, and are known as the *cysticercus telæ cellulosæ*. In these situations they can develop no further, and in course of time perish; but when the flesh containing them is eaten by man, or a carnivorous animal, the head and neck are extended from the globular cyst, the cyst is dissolved in the stomach of the host, the head attaches itself by its suckers to the alimentary mucous membrane, and the segments of tænia successively grow upon it until a complete tapeworm (*strobila*) is formed.

The **Symptoms** and **Treatment** are the same as those of Tænia mediocanellata.

TÆNIA MEDIOCANELLATA VEL SAGINATA

This is the tapeworm most commonly met with in England. In addition to the water-vascular canals present in the Tænia solium, this worm possesses a third, occupying the middle line. Its head is provided with four suckers, but is without proboscis or hooklets. In length the animals may attain five or six yards, and the segments number from 1200 to 1300. The complete development of the sexual organs occurs about the 600th segment; the last 150 to 200 are ripe proglottides. The mature segment measures three-quarters of an inch in length, by one-third in width, and the uterus extends the whole length of the segment, giving off on each side from twenty-five to thirty lateral branches, which divide at their extremities. The ova are only a little larger than those of Tænia solium, and have the same shape. The cysticercus is commonly found in beef or veal, and not in pork. The mature segments frequently find their way out of the anus, independent of the act of defæcation.

Symptoms.—The presence of the worm may cause no symptoms at all, and it may only be recognised by the discovery of segments in the motions. Sometimes disagreeable sensations in the abdomen are described, or gnawing or colicky pains, irregularity of the bowels, and deficient or voracious appetite; itching at the nose or at the anus, salivation, and vomiting also occur. More remote symptoms are giddiness, faintness, and languor; headache, mental disturbance, depression, and even fits, either hysterical or epileptic in character. These are more likely to be aggravated in persons of hypochondriacal or hysterical tendencies. It is obvious that there is nothing pathognomonic in these symptoms, they can only give rise to a suspicion, which must be confirmed by the appearance of the segments. These the physician should himself

see, since they may be simulated by fragments of mucus or half-digested food. It may be desirable in some cases to give a purgative to bring away segments.

Treatment.—This should never be undertaken unless the presence of a tapeworm is absolutely certain. The method of treatment is the administration of a drug, which is fatal to the worm, and the subsequent removal of the worm by a purgative. In order that the former drug, or anthelmintic, may come into full contact with the worm it is desirable to have the intestines as empty as possible. In most cases it is sufficient for the patient to have no food after six or seven in the evening, and to take the anthelmintic before breakfast the next morning. If the bowels have been previously confined, they may be cleared by a dose of castor oil the day before the morning dose. In either case, the anthelmintic should be followed in three or four hours by a dose of castor oil or compound rhubarb powder. Several drugs will destroy the tapeworm; the most commonly used in England is male fern, of which the liquid extract may be given in a dose of 1 to 1½ drachms suspended in mucilage. Other remedies are kousso in the form of infusion; oil of turpentine from ½ to 2 ounces, which should be followed by a purgative to ensure its not being absorbed; decoction of pomegranate root bark in three or four doses of 1 or 2 ounces each every half-hour; tannate of pelletierine, 8 to 12 grains in capsule; and kamala powder, 1 to 3 drachms in wine or water.

The dead worm must be looked for in the motions which follow this treatment by mixing them with water, stirring, and pouring off the upper portions from time to time. The cure cannot be considered complete unless the head of the worm is found; for the worm may break at the neck, and if the head remains attached to the bowel, it will give rise to fresh segments, and ultimately to a complete tapeworm. In this case, a period of almost exactly three months elapses before the mature segments again appear in the fæces; and as it is obvious that the head may elude even a very careful search, it is quite as well not to repeat the treatment forthwith, but to wait until the reappearance of segments conclusively shows that there is still a worm in the bowel.

BOTHRIOCEPHALUS LATUS

This worm is much larger and longer than either of the preceding, measuring from seventeen to twenty-six feet in length. The head is elongated, and presents only two suckers in the form of long grooves (*βόθρος*, a trench). The segments are about 3000 in number; they measure in the middle half an inch broad, and only one-seventh of an inch in length; but lower down they become more square in shape. The uterus is unbranched, but is bent several times upon itself. The ova measure 0·7 mm. in length, and have a lid at one end. The portions of the tapeworm that are detached are often several feet in length. The life-history is similar to that of the

tænia, but the cysticerci inhabit fish instead of herbivorous animals. Thus, the ova are developed only in fresh water, and form embryos, which are provided with six hooklets and numerous cilia; by means of the last they swim about freely. They are then swallowed by fishes, especially by pike and eel-pouts, and in their muscles and internal organs take on the form of cysticerci. The Bothriocephalus latus is found especially in Switzerland and Central Europe.

Symptoms.—In contrast with the tæniæ, Bothriocephalus latus nearly always produces an *anæmia*, which may present all the blood characteristics of pernicious anæmia. Thus there is a great diminution of red blood corpuscles, a colour-index above unity, and the presence of poikilocytes, megaloblasts, normoblasts, and corpuscles showing polychromasia and basophile stippling. The anæmia is the result of absorption of the products of decomposition of the parasite, producing hæmolysis, and acting upon the bone-marrow.

Treatment.—This is the same as is employed for the tæniæ.

ASCARIS LUMBRICOIDES

Anatomy.—In shape and general appearance this resembles the ordinary garden worm (lumbricus); it is pink, cylindrical, and tapering at each end. The mouth is at one extremity, and is surrounded by three tubercles or lips provided with fine teeth; and it communicates with an intestine running the whole length of the animal. The male ascaris is about ten inches in length; it is seldom met with. The female is from twelve to sixteen inches in length, and it has been estimated that the organs of generation can contain at one time sixty millions of ova. These measure 70μ in length by 60μ in breadth. They have a dirty brown colour and are nodulated on the surface from the presence of an albuminous substance deposited outside the shell. They are found in the fæces of the host. There is still much doubt as to the manner in which the ascaris develops after the formation and escape of the embryo.

These worms inhabit the small intestine, where they may be passed *per anum*, or they may reach the stomach and be vomited. They have a curious tendency to insert themselves into apertures, or ring-like bodies, that may have been swallowed, such as the shanks of buttons; they have been found blocking the common bile-duct, the glottis, or the nasal passages; and occasionally they occur in abscesses in the groin or some part of the abdomen, about which it is not always easy to say whether the inflammation has been set up by the worm or by some other cause. The number of ascarides which may be present in the same individual is very variable; there may be only one, often there are only two or three, but sometimes they are in great number.

Symptoms.—These are not very different from those set up by tapeworms. On the other hand, there may be none. Nausea, foul breath, irregular appetite, itching of the nose, or abdominal pain may be present. In other cases there may be reflex symptoms,

such as fits, choreic or convulsive movements, or mental disturbance. But the parasite may lead to more serious troubles, such as jaundice, by obstructing the bile-duct, or suffocation, by entering into the larynx; and occasionally the worms have formed a convoluted mass large enough to cause intestinal obstruction.

Diagnosis.—Here, also, the diagnosis depends on the appearance of the worm or the discovery of its ova in the fæces. If a worm is discharged through the anus, or by vomiting, it is as well to treat the case as if others existed. Even when several have been expelled by treatment there may be others left behind; and this may be shown by the detection of the ova in the fæces. As specimens of the garden worm may be brought to the physician and passed off as ascarides, the following differences should be noted: the earthworm is redder in colour, it is less tapering at its extremities, it is provided with bristles along the sides to aid its progression, and its mouth is a short transverse fissure on the under surface of the rounded head.

Treatment.—The best treatment for the Ascaris lumbricoides is the administration internally of santonin, the active principle of wormseed (santonica). It is tasteless, and can be taken as a powder mixed with sugar, or simply placed on bread and butter, or suspended in milk. The dose for a child is 2 or 3 grains, for an adult from 4 to 6 grains; it should be taken on three or four successive mornings, and followed by a calomel purge, or a dose of compound rhubarb powder. Santonin sometimes affects the vision, so that objects appear green, yellow, or blue; or it may cause tenesmus, or hæmorrhage from the bowels. Severe nervous symptoms, convulsions, and collapse have followed large doses. The urine is always coloured bright yellow, and is then turned red by the addition of an alkali.

OXYURIS VERMICULARIS

This, the threadworm, is very much smaller than the preceding. The female is about half an inch long, the male only one-sixth of an inch. It has a mouth and a complete alimentary canal, and the uterus of the female develops an enormous quantity of ova, which measure 50μ by 23μ, are elongated, curved, provided with an operculum, and contain the embryo already formed. The adult thread-worms occur only in the large intestine, to a great extent in the rectum, where they are often matted together in balls; but also in the cæcum. There is, however, no new generation *in situ*, but the ova must be first taken into the stomach of the host, whether it be the same individual or another; and infection probably takes place from child to child, the ova drying on the clothes or on the skin and hair about the anus, and being conveyed by the fingers in scratching or otherwise. And as the ova must also be present in the fæces of those affected, they may sometimes, from imperfect sanitary arrangements, get into drinking water, and be carried thereby to other people. The embryos are then set free

in the upper part of the alimentary canal, and reach their full development in the cæcum, whence they generally move down into the rectum.

Symptoms.—These are mainly local, and due to the presence of the parasite in the rectum. The chief symptom is heat or itching at the anus, and this is worse at night, when the patient gets into bed, or shortly before this. There may be at the same time irritability of the bladder, with frequent micturition; or tenesmus, prolapsus ani, or excessive secretion of mucus; and in girls the worms may creep into the vagina, and set up irritation at the vulva, or cause a vaginal discharge.

Treatment.—The use of purgatives, such as calomel, scammony, and jalap, will, of course, bring away some worms; but the object of treatment should be to kill them *in situ*. So far as the rectum and lower bowel are concerned, this may be effected by astringent enemata, such as infusion of quassia, with or without some solution of perchloride of iron, solution of alum (7 or 10 grains to the ounce), of tannin, of common salt, or of lime. The rectum should be cleared by a warm-water enema, and 5 or 6 ounces of the astringent should be injected, and kept in for some time. This should be repeated two or three times a week for two or three weeks. To destroy the worms resident in the cæcum it has been recommended to give saline purges frequently, or large doses of infusion of gentian or quassia internally. The itching at the anus is relieved by the application of unguentum hydrargyri. The constant application of a mercurial ointment (ung. hyd. nit.) for six weeks has also been recommended to prevent re-infection by the fresh deposited ova.

TRICHOCEPHALUS DISPAR

This nematode worm measures one and a half to two inches; in its anterior two-thirds it is extremely fine like a thread or hair, but the posterior third is thicker. It inhabits the cæcum, but rarely gives rise to clinical symptoms. The ova, which may be found in the fæces, have a long oval shape, and measure 50μ in length by 23μ in breadth.

ANKYLOSTOMA DUODENALE

This parasite, also known as *Sclerostomum duodenale*, and *Dochmius*, or *Strongylus duodenalis*, is a small nematode worm, which attaches itself in great numbers to the mucous membrane of the duodenum and jejunum, and causes among other symptoms a high degree of anæmia. The disease occurs in many parts of the world. The so-called "Egyptian chlorosis" is due to this parasite; it occurs in Italy among workers in furnaces, and in Westphalia among miners, and it was the cause of the numerous cases of anæmia occurring in 1880 among the labourers in the St. Gothard tunnel. In 1902 it was discovered in England, in a Cornish tin-mine, by Drs. Boycott

and Haldane; though it is probable that the disease had existed there since 1894.

Precisely similar symptoms occur in the Southern United States of America and in Porto Rico. But the worms found in these cases mostly belong to a different species, called *Uncinaria*, or *Necator Americanus*, or *Ankylostoma Americanum*: and the disease is known there as *uncinariasis* or *hookworm disease*. The only material difference between the two species is in the structure of the anterior extremity, or mouth.

The female of the ankylostoma duodenale is half an inch in length, and the male about one-third of an inch. The ova, which can be detected in the fæces, are oval in shape and measure from 50μ to 60μ in length, by 30μ to 40μ in breadth. The egg-shell is smooth, thin, and apparent as a single line only, transparent, and showing from 4 to 16 cells in the interior. The ova are hatched outside the human body and produce larvæ, which after growing to a certain size are surrounded by a hard coating, and are said to be encapsuled. In this condition they can neither grow larger nor reproduce themselves, until they enter the body of their human host. This they do, as was first shown by Looss of Cairo, by penetrating the skin, and rarely by being introduced into the mouth. They are found in the subcutaneous connective tissue, and Looss traced them into the lymphatics, thence into the lungs, whence it appears they reach the bronchi, are coughed up through the larynx, and hence by the pharynx reach the stomach and intestines. The heat and moisture of mines, and other places where they are prevalent, are favourable to their growth, and the naked bodies of the workers give ample opportunities for direct invasion. In Porto Rico the A. Americanum infests the coffee plantations, where the men work in a moist infected earth with naked feet and legs. The absence of proper arrangements for the disposal of sewage contributes in all these instances to the multiplication of the worms.

Symptoms.—A few worms may be present in the bowel, and ova may be found in the fæces, without any other sign or symptom to indicate their presence. But in larger numbers they produce a severe *anæmia*, and where the larvæ are constantly being brought into contact with the skin, they produce a characteristic *eruption*. The eruption is called "bunches" by the Cornish miners: it occurs commonly on the forearms and hands, and consists of papules, pustules or furuncles, and urticarial wheals. The anæmia is shown by a gradually increasing pallor of the face, lips, conjunctivæ, and body generally; puffiness of the face and feet; feebleness and lassitude, with quick, small pulse, palpitation, dyspnœa, and deranged digestion. The anæmia is of a severe chlorotic type (*see* Anæmia), with a diminution of the hæmoglobin to 40 per cent., a low colour-index, a large increase in the total volume of the blood (hydræmic plethora), a varying increase in the leucocytes, and a marked relative and absolute increase in the eosinophile cells, which may reach from 30 to 50 per cent. of the leucocytes (Boycott and

Haldane). The anæmia is probably not due to direct abstraction of blood from the bowel by the worms: but the tissue of the mucous membrane is found to be degenerated and eroded at the seat of their attachment, and the altered state of the blood is possibly due either to absorption of toxins, or to defective secretions in consequence.

Prognosis.—The anæmia can be promptly cured, the patient restored to practical health, and the majority of the worms can be expelled from the intestine; but it is extremely difficult to get rid of the last few worms, and ova may be found in the fæces months and years after recovery.

Diagnosis.—When the disease occurs as an epidemic disease among groups of workers underground its recognition is of course easy. The cutaneous eruption, or the dyspnœa, but especially the anæmia, brings the patient under observation: but in Egypt the diagnosis is complicated by the prevalence of another parasitic disease, namely, bilharziosis, which also causes anæmia. In any case, however, the character of the blood-count will be a guide to the kind of anæmia, and the presence of the worm can be determined by finding the ova in the fæces. A small piece of fæces may be mixed with water on a slide and examined with the microscope; or it may be shaken up with water in a test-tube, when the eggs will fall to the bottom, and the supernatant fluid may be decanted. If the eggs are in small number, they may be separated by the use of solutions of calcium chloride. A portion of fæces is washed first with a 12 per cent. solution of $CaCl_2$ of sp. gr. 1050, so that light materials are separated; and afterwards a 55 per cent. solution of $CaCl_2$ of sp. gr. 1250 is added. The eggs, having a sp. gr. of 1100, float to the surface. A certain method of diagnosis is by cultivation; the eggs are incubated at from 30° to 36° C. for from five to seven days.

Treatment.—The anthelmintics commonly employed have been thymol, eucalyptus oil, β-naphthol, and oil of male fern. The last appears to be useless. The first two are the most efficacious. Thymol should be given in half-drachm doses in capsule, and no alcohol or oil should be taken for some time before and after. The bowels should be cleared by a dose of sodium sulphate given at night, one dose of thymol should be given the next morning early and then two hours later, and a second dose of sodium sulphate in the evening. Eucalyptus oil may be given in a dose of 45 minims with 45 minims of spiritus chloroformi; and repeated once or twice. Iron and arsenic may be given for some weeks afterwards till the anæmia is cured.

DISEASES OF THE LIVER

The liver occupies the right hypochondriac region, under the ribs, and stretches across the upper part of the epigastrium. Normally it can scarcely be felt even in the latter situation, and there only when the abdominal parietes are very thin. Percussion gives dulness (*hepatic dulness*) in the mammary line, from the upper border of the sixth rib to the costal margin; in the middle line there is very slight loss of resonance for one and a half or two inches from the base of the ensiform appendix, where the thin left lobe lies over the stomach. When the abdominal parietes are thin the edge of the liver may be perceptible to sight during deep inspiration, as the organ descends for one and a half inches, and the percussion-dulness shifts to a corresponding extent. In the axillary line the hepatic dulness begins at the eighth rib, and at the tenth rib in the line of the scapular angle.

In disease the organ is often enlarged. It then, as a rule, projects below the costal margin, and can be felt as a definitely resisting mass, different from the supple part of the abdomen below it and in the left hypochondrium. It may reach to the level of the umbilicus, or much lower, its lower margin extending across the abdomen from the right flank to the left costal margin. It presents different degrees of consistence, and alterations of surface, according to the disease affecting it; and the dulness extends to a corresponding degree down into the abdomen, since the organ always lies in front of the hollow viscera, unless there is liquid in the peritoneal cavity, which will enable the liver to fall away from the anterior parietes. From the presence of the intestines behind it, the dulness is not so complete near the free edge as it is higher up, and the percussion must be lightly performed in order to localise it exactly. In the presence of ascites, percussion is useless as a test of the size of the liver.

The liver only encroaches on the chest when the enlargement is (1) localised rather than general, such as that due to cancer, hydatid, or abscess; and in these cases the ribs may be bulged outwards so as to enlarge the right costal angle; or (2) when the liver is itself pushed up by something below.

Apparent enlargement of the liver arises from tight-lacing, and from tumours or pleuritic fluid in the chest. The former elongates the organ vertically (*see* p. 784); in the latter the whole liver is displaced. Displacement of the liver downwards, or proptosis, occurs also as a part of *Glénard's disease* (*see* p. 821).

In atrophy of the liver there is a diminution of the percussion-dulness, but a similar diminution may be caused by the encroachment of intestines distended with air.

A distended gall-bladder may be felt as a globular prominence at the lower border of the hepatic dulness, in the mammary line.

Two common results of hepatic disease are jaundice and ascites, and these conditions will be discussed before the special diseases of the liver are described.

JAUNDICE

By the term *jaundice* (from *jaune*, yellow), or *icterus*, is meant a yellow discoloration of the skin and other parts by bile-pigment circulating in the blood. In ordinary cases, such as those which arise from obstruction of the common bile-duct, the skin has a more or less deep yellow tinge, the conjunctivæ are yellow, and the visible mucous membranes have their natural red colour obviously modified by the yellow tint. In long-standing cases the colour of the skin becomes deeper, and finally of a greenish- or olive-brown tint. This, formerly distinguished by the name of *black jaundice*, is due, no doubt, to the gradual conversion by oxidation in the skin of bilirubin, the yellow pigment of the bile, into biliverdin. The yellow colour must be distinguished from other changes of colour in disease, such as the yellowish tinge of cases of chlorosis and of pernicious anæmia, the sallow tint of malarious cachexia, and the brown colour of Addison's disease. The colour can be generally well recognised in the conjunctiva, but in some people small masses of subconjunctival fat give a tint which is not very unlike it.

The colour of the *urine* is at the same time altered, from the presence of the biliary pigment. In small quantity this gives it a bright saffron colour, which is best seen in any froth which may form on the surface; if there is more the urine becomes brownish-yellow, or yellowish-brown, or even dark brown like porter. If linen or paper is dipped in the urine, it is stained bright yellow; but the presence of bile-pigment can be more certainly proved by the application of chemical tests which will be mentioned presently. Of the other secretions of the body the majority are not discoloured: occasionally the sweat is tinged yellow, and sometimes the milk of nursing women. The tears, saliva, gastric juice, and intestinal secretions are unaffected; but the secretions from the mucous membranes, when diseased, and more frequently morbid effusions from serous membranes, may contain some biliary pigment. At the commencement and at the end of an attack of jaundice, the urine often contains urobilin, a derivative of bile-pigment, but none of the bile-pigment itself.

In most cases of jaundice the *fæces* are altered in colour, becoming whitish or clay-coloured; this is due to the absence from them of urobilin, in those cases of jaundice where the bile is unable, from obstruction of the ducts, to find its way into the duodenum. The absence of bile from the chyme may have other effects, since this secretion is known to have some share in the digestion of fat, and is thought to have a power of preventing putrefaction in the

intestinal contents, and to stimulate the muscular fibres of the intestinal wall. Accordingly, it is common to find fat in the stools; they are sometimes offensive, and constipation is frequent, though by no means invariable. When diarrhœa occurs, it has been attributed to the irritation of the putrescent fæces.

Other symptoms are often present in jaundice, which are no doubt due to the circulation in the blood of the constituents of the bile. Occasionally the *pulse* becomes slowed to fifty or forty per minute. This is probably owing to the action of the bile-acids upon the cardiac ganglia. It is most common in cases of catarrhal jaundice. *Itching* occurs, especially when the jaundice is due to obstruction of the bile-ducts; and it may be so intense that sleep is rendered impossible, and blood-crusts, papules, or wheals of urticaria are produced by the incessant scratching. The cause of the itching is uncertain; it seems not to be due to the bile-pigment alone, for it has been noticed in cases some time before the jaundice appeared. A disease of the skin named *Xanthelasma* or *Xanthoma* occurs in some cases of long-standing chronic jaundice (*see* Diseases of the Skin).

Some patients have a bitter taste in the mouth, and digestive disturbances are frequent. Hæmorrhages take place under the skin or from the mucous surfaces, and the bleeding from wounds is not readily checked; the coagulation-time of the blood is prolonged. In some cases, serious cerebral symptoms arise, such as delirium, convulsions, and coma; but these are probably always due to the presence in the blood of other poisons than those contained in the bile.

When in a case of jaundice the common bile-duct is not blocked, and obstruction, if present at all, involves only the minute bile-ducts, and perhaps not the whole number of them, the icterus is often wanting in some of the features described above. Thus it is less deep, the fæces are naturally coloured, the urine may contain urobilin, and no bile-pigment, and bradycardia and pruritus are absent.

Tests for Bile-Pigment in the Urine.—The essential feature of these tests is the production of a green colour by the oxidation of yellow bilirubin into green biliverdin: in some processes other tints are temporarily developed. The most commonly employed is Gmelin's. A few drops of urine are placed upon a white plate, and a little strong nitric acid is dropped close by, and then the two fluids are gently run into one another. At the line of contact, the colour of the urine changes, becoming green, blue, violet, red, and lastly yellow or brown. The urine may be slowly poured by means of a pipette on to the surface of nitric acid, placed in a test-tube, when a similar result will be obtained. If nitric acid be added to the diluted urine in the test-tube, the mixture will turn green.

If the quantity in the urine is very small, calcium chloride solution should be added to the urine, and then solution of sodium carbonate. The precipitate is filtered off, washed with a little

water, and redissolved in dilute hydrochloric acid. To this Gmelin's test may be applied (Ryffel).

The oxidation may be effected by iodine: if a few drops of a solution of iodine be dropped on to the surface of the urine in a test-tube, the urine becomes of a deep green colour (Maréchal's test).

Test for Bile-Acids in the Urine.—The special significance once attached to the presence of bile-acids in the urine, apart from the bile-pigment, is not now allowed: and in any case the tests for them are difficult and untrustworthy. Pettenkofer's test is the production of a purple colour on the addition of sugar and sulphuric acid; but the bile-acids must first be separated from the urine. In Haycraft's test powdered commercial sulphur is dusted on to the surface of the urine: it sinks more readily when bile-acids are present.

Explanation of Jaundice.—It is not difficult to explain the occurrence of jaundice in the cases in which the outflow of the bile is prevented by any stricture or obstruction of the common bile-duct. The bile distends the gall-bladder and the bile-ducts, and then passes into the lymphatics and blood-vessels, circulates in the latter, and gives the characteristic tinge to the skin and other parts. An interesting fact in the secretion of the bile makes it likely that a complete obstruction is not necessary—that is, that the bile is secreted under very low pressure, such that in guinea-pigs a pressure of twenty centimetres of water will force the secreted bile back into the circulation. Where there is a complete obstruction, as from a gall-stone in the common duct, or a cancerous tumour pressing upon it, the bile is unable to reach the intestines, and the fæces, as already stated, are white or clay-coloured. This is called *obstructive* jaundice. If the obstruction is removed—as, for instance, by the gall-stone passing at length into the duodenum—bile again flows into the intestine, the fæces become dark, the urine acquires a normal colour, and the skin more slowly loses its jaundiced hue.

The explanation of those cases of jaundice in which the bile-ducts are not obviously blocked is more difficult; but it is also based upon obstruction, which affects the minute ducts throughout the liver instead of the main bile-duct in the hilum. Thus the structural changes which occur in the substance of the liver in cirrhosis may be supposed to affect the viability of the minute ducts. But some other explanation is needed where the liver is to the naked eye normal.

If the substance named *toluylendiamine* is taken internally, it causes jaundice; and this it appears to do by destroying the blood-corpuscles and liberating hæmoglobin. The hæmoglobin being carried to the liver increases the quantity of bilirubin, and the bile thereupon secreted contains much bile-pigment, but little bile-acid. Though at first secreted in quantity, it soon becomes thick, viscid, and tenacious, and so increases the pressure in the bile-ducts that the secreted bile is absorbed by the lymphatics and carried into the circulation and jaundice results.

This destruction of the red blood corpuscles, or *hæmolysis*, appears

to be the antecedent of the jaundice in some non-obstructive forms, such as those called congenital acholuric jaundice, congenital familial cholæmia, and icterus neonatorum. The jaundice in such cases is classed as *hæmolytic jaundice:* but it is still open to question whether the jaundice is due to a catarrh of the small bile-ducts caused by the poison which produces the hæmolysis, or by the excess of blood-pigment with which the liver has to deal, or to increased viscidity of the bile allowing its easier passage into the lymphatics.

The jaundice which occurs in poisoning by phosphorus, arsenic, and antimony, and that which arises in association with numerous infectious diseases, enteric fever, relapsing fever, yellow fever, and others, are probably to be explained in a similar way.

Thus, nearly all forms of jaundice arise by absorption of bile secreted in the liver and delayed in the ducts either by mechanical obstruction of the hepatic or common bile-duct, or by actual or relative obstruction in a number of small ducts. The actual obstruction is narrowing of their channels—the relative obstruction, such an increase or alteration of the secretion as to make it flow too slowly through them, or such alteration in the relative pressure of bile in the small ducts and fluid in the vascular system as will drive the bile-pigment into the latter.

Causes of Jaundice.—These are·

Obstruction of the Larger Bile-ducts: (1) Gall-stones and inspissated bile, hydatids, distomata, and foreign bodies from the intestinal canal, including *ascaris lumbricoides.* (2) Stricture or obliteration of the duct from congenital defect, from perihepatitis, or from former ulceration of the duodenum or of the bile-duct itself; catarrhal or inflammatory swelling of the wall of the bile-duct; spasm of the duct. (3) Compression by tumours, abscess or hydatid of the liver, and by glands in the portal fissure; by tumours of the stomach, colon, head of the pancreas, kidneys, omentum, ovaries, or uterus; by an abdominal aneurysm, accumulated fæces, or pregnant uterus.

Probable Obstruction of the Minute Ducts: (1) By tissue changes; various forms of cirrhosis. (2) By increased viscidity of the bile in the minute bile-ducts, the result of catarrh of the ducts; poisons such as phosphorus, arsenic, antimony, mercury, and others; snake poison; infectious diseases, namely, typhus, enteric fever, relapsing fever, yellow fever, malarial fevers, scarlatina, pyæmia, and possibly acute pneumonia; different forms of acute, febrile, malignant, and infectious jaundice, acute yellow atrophy, and Weil's disease; acute and chronic congestion of the liver; hæmolytic jaundice.

The discrimination of the various forms of jaundice must depend on a consideration of the diseases to be presently described; but it may here be pointed out that the most common forms in English practice are (1) catarrhal jaundice, and those associated with (2) gall-stones, with (3) cancer of the liver and portal glands, and cancer and chronic inflammation of the head of the pancreas, and with (4) cirrhosis.

ICTERUS NEONATORUM

Jaundice in infants is often a temporary condition which passes off in a few days. Formerly attributed to mechanical causes, it is now ascribed to hæmolysis of doubtful origin. For two or three days the skin presents a deep red colour, and the urine is highly coloured: then the skin becomes jaundiced, the urine contains urobilin, and the blood-serum is stained with bile-pigment.

Less frequent and more serious causes are congenital obstruction of the bile-duct, septic infection through the umbilical vein, and syphilitic disease of the liver.

ASCITES

By this term is meant the presence of serous fluid in the peritoneal cavity. Like other effusions into the serous cavities, it is commonly alkaline, of a pale straw colour, of specific gravity 1015 to 1018, highly albuminous, and containing chlorides. It arises (1) from obstruction of the portal circulation, either in the trunk of the portal vein, or in its distribution in the liver; (2) as a result of diseases of the peritoneum; and (3) as a part of the general dropsy of renal disease.

The portal vein trunk may be obstructed by the pressure of tumours and enlarged glands in the portal fissure, by cancer, abscess, or hydatid in the liver itself, and by coagulation of blood in its interior (*thrombosis, pylephlebitis*). In the liver the chief cause of portal obstruction is the compression of the interlobular veins by the fibrous overgrowth of cirrhosis. It is thought by some that portal obstruction is not an adequate cause of ascites, which they attribute to toxins produced in the diseased liver, or absorbed from the intestine and undestroyed by the liver. Another cause of portal obstruction is perihepatitis. A third kind of obstruction is formed by the different forms of cardiac and lung disease, in which the right side of the heart is dilated, and the passage of the blood through the chest is impeded (*see* Diseases of the Valves, *and* Emphysema of the Lungs).

The peritoneal diseases causing ascites are—acute and chronic peritonitis, tubercular peritonitis, and cancer of the peritoneum.

In Bright's disease, the peritoneum is the seat of effusion in common with the other serous cavities.

Ascitic fluid is in rare cases opalescent and milky instead of a clear serum. One variety of this is *chylous ascites*, due to chyle which has entered the peritoneum from ruptured lacteal vessels, or has transuded on account of pressure on the thoracic duct: it contains fat droplets, lymphocytes, and sugar (*see* Filariasis). A second variety is *chyliform ascites*, in some cases of which the turbidity is due to fatty degeneration of epithelial cells in the course of chronic peritonitis (*adipose ascites*), and in others to suspended proteid granules, compounds of lecithin and globulin.

The **Physical Signs** of ascites must be carefully attended to, as it is not impossible to confound it with other conditions. The abdomen, of course, enlarges, and in the early stages of a considerable ascites it is generally tense, and the form tends to be globular, with a decided prominence in a forward direction. Later the walls of the abdomen become stretched, and as the patient lies in bed the fluid gravitates backwards, and gives a broader and flatter shape to the belly. The liquid then poured out may amount to three, four, or five gallons, and the abdomen becomes proportionately enlarged so that it may measure from forty to forty-two inches or more in circumference. The presence of fluid is detected by three methods of examination—*percussion*, *fluctuation*, and *displacement*.

Percussion.—Normally, the surface of the abdomen is resonant, from the air contained in the stomach and intestines; but when fluid is poured out, this collects at first in the flanks and hypogastric region, so as to give a dull note to percussion in these parts, while the centre of the abdomen remains resonant.

As the fluid increases, the dulness encroaches more and more from the sides and hypogastrium upon the centre, and at length only a limited area remains resonant—namely, that which includes the umbilical and the left hypochondriac regions. If, in either of these stages, the patient be turned upon one side and again percussed, it will be found that the anterior and central regions have become dull, and the flank, which is now uppermost, gives a resonant note. This is due to the gravitation of the fluid to the lowest part, and the floating of the air-containing bowel to the highest; and this occurrence is the most conclusive proof of the presence of fluid in the peritoneum. Occasionally, however, the abdomen is entirely dull, when the mesentery is so short, or the fluid so abundant, or the viscera are matted down by chronic peritonitis in such a way, that the intestines cannot float to the uppermost part. Then also this test by change of position fails to give the desired information.

Fluctuation is obtained by laying one hand on one side of the abdomen, and sharply tapping or flipping the other side with the finger. The applied hand then feels the transmission of a wave across the abdomen. This is a less certain sign than the former. Very fat abdominal walls may transmit a wave without the presence of fluid, and to prevent this, the edge of the hand, or of a book or card, should be pressed on the centre of the abdomen while fluctuation is tried.

The method of *displacement* has only a limited application, but it provides in some cases earlier evidence of ascites than either percussion or fluctuation. If in a case of ascites the liver is enlarged, it sinks in the fluid, and a small quantity of fluid lies between its anterior surface and the abdominal wall. By placing the fingers on the abdomen at this spot, and suddenly pressing them in, the fluid is displaced, and the surface of the liver may be felt. This is a proof of the presence of fluid; since, if there were none, the liver would be in close apposition with the anterior abdominal wall.

Ascites is, however, sometimes simulated by one or other of the different kinds of cyst which may occur in connection with the abdominal or pelvic viscera, by a pregnant uterus, or by a distended urinary bladder. These cysts are ovarian, parovarian, hydatid, or renal cysts. They are excluded if the percussion test is successful; on the other hand, they may give the fluctuation test; and if the whole surface is dull there may be some difficulty in distinguishing between one of those and an ascites in which the intestines are bound down. An operation for ovariotomy has several times been attempted, at which the case has been proved to be one of ascites. Ovarian dropsy is chiefly distinguished by the abdomen being dull in front and resonant in the flanks, into which position the intestines are pressed by the cyst; and by the swelling at least beginning on one side, though it is afterwards central. Not infrequently also the outline of the cyst can be recognised at the uppermost part, especially if looked for during the movements of respiration. Some of the measurements of the abdomen are different in the two cases; thus, normally, and in ascites, the umbilicus is about an inch nearer to the pubes than to the sternum; in ovarian cysts this ratio is often reversed. In the latter, also, the distance from the umbilicus to the crest of the ilium may be greater on the side of the diseased ovary, and the greatest girth of the abdomen is an inch or two below the umbilicus; whereas in ascites it is at the umbilicus or a little above it. The fluid withdrawn by tapping an ovarian cyst is usually glairy, and gray or yellowish-gray, or grumous, brown or chocolate-coloured from containing altered blood; a parovarian cyst yields a clear watery fluid containing only a trace of albumin and a little saline matter; and hydatid fluid is somewhat similar (*see* p. 790).

CIRCULATORY CHANGES IN THE LIVER

ANÆMIA

This does not occur as a separate affection. The liver suffers with other organs in conditions of general anæmia; fatty, lardaceous, and cirrhotic livers doubtless contain less blood than normal livers. It is not uncommon to find at an autopsy patches of a pale colour due to local anæmia, but they have no clinical significance, and are possibly, indeed, of *post-mortem* occurrence.

PASSIVE CONGESTION

The *nutmeg liver*, which has been already described (*see* p. 591) as one of the results of valvular disease of the heart, is an extreme form of passive congestion. In earlier stages of the same change the organ is simply engorged, being larger than normal, and dark red in colour, with obvious distension of the intra-lobular veins. It causes fulness and discomfort in the right hypochondrium, and

ultimately ascites and slight jaundice. Hepatic pulsation is occasionally associated with it.

ACTIVE CONGESTION

Active congestion of the liver no doubt arises in the course of various febrile illnesses from the circulation of toxins, and it must form a part of acute inflammation of the organ. It is common also to regard as due to active congestion the symptoms which occur in those who have attained thirty-five or forty years of age, who live sedentary lives, and eat and drink freely. These are a sense of weight, oppression, or fulness in the right hypochondrium, increased by pressure, by tight clothes, or by lying on the left side; pain in the right shoulder, furred tongue, nausea or sickness; slight jaundice, constipation, and the passage of high-coloured, scanty urine, depositing urates. On examination, the hepatic dulness may be found reaching below the costal margin, or the edge of the organ may be felt. In addition the patient suffers from a bitter taste in the mouth; aching pains or severe cramps in the limbs; lassitude, drowsiness, headache, and giddiness; grinding of the teeth; sleeplessness; palpitation, fluttering, and intermittent action of the heart. The urine is concentrated, high-coloured, and deposits urates; it contains an excess of urobilin. The congestion of the liver is probably set up by the conditions of gastric or intestinal catarrh which occur in such patients. Some of the symptoms are directly due to the same causes. No doubt toxic agents are in operation, especially when one remembers the frequency of hepatic congestion verging on inflammation, which occurs in the tropics, both in connection with excesses and with malaria and dysentery.

Treatment.—The patient should be kept at rest, on a milk diet, and poultices should be applied. If the pain is great, ice may sometimes be applied, or dry cupping, or leeches may be used. Internally small doses of calomel may be given, or pil. hydrargyri (2 to 4 grains) with pil. coloc. co. (3 to 4 grains), followed by mist. sennæ co. or sulphate of sodium or magnesium. The bowels may further be kept active by occasional saline purges, or the daily use of Püllna, Friedrichshall, Carlsbad, or Hunyàdi János waters. The later treatment consists in the careful regulation of the diet, taking active exercise, and the use of mercurial and other purges. Alcohol should be limited to a little claret or Rhine wine with dinner; and meat, spiced and made dishes, should be taken only in small quantities.

ACUTE HEPATITIS

Acute inflammation of the liver is not a common affection in England, and in tropical countries it often ends in abscess. But it may terminate by resolution in a week or ten days. Its symptoms are essentially the same as those which precede the development of suppuration (*see* Abscess of the Liver).

ABSCESS OF THE LIVER

Pathology.—Abscesses of the liver arise under a variety of conditions, but what is common to nearly all of them, except injury, is the introduction of some septic agent by one of three channels: the hepatic artery, the portal vein, or the bile-ducts.

In the first case they form part of a general pyæmia, such as results from wound or injury in any part of the body, but especially injuries to the head; they are small in size, and numerous, or at least multiple. They are known as *pyæmic abscesses.*

The portal vein is responsible for a still larger number of cases, the septic agents being carried from lesions within the portal vein area, such as gastric ulcer, appendicitis, pelvic suppurations, and especially ulcerative lesions of the intestine, including tropical dysentery. The abscesses may be single, few, or multiple; and when they are multiple the condition may be spoken of as *portal pyæmia.* Sometimes the portal vein and its branches are filled with broken-down purulent clot, and the walls of the veins are inflamed, constituting *suppurative pylephlebitis.* From this group of cases it is difficult to exclude the well-known *tropical abscess,* which is constantly seen in association with dysentery, but which is believed by some to arise independently. In support of this last view it is stated that cases of hepatic abscess occur without any clinical or *post-mortem* evidence of dysentery, and that the symptoms of dysentery sometimes follow hepatic abscess instead of preceding it. The frequency with which tropical abscess is solitary and of large size lends colour to the view that it is different from the multiple small abscesses of portal pyæmia. But multiple abscesses sometimes occur in dysentery; and out of the tropics a large single abscess sometimes follows an ulcerative lesion of the intestine which is not dysenteric. Moreover, the discovery of the *amœba coli* in abscesses of the liver associated with dysentery due to the same organism shows that in some cases there must be a causative connection between the two lesions.

Invasion by the bile-ducts is chiefly effected in consequence of gall-stones ulcerating into them (*suppurative cholangitis*).

The difference to be noticed in the symptoms in various cases of hepatic abscess makes it desirable to consider separately the multiple abscesses, which are generally pyæmic, and the large solitary abscess, which is so often tropical.

MULTIPLE ABSCESSES

The abscesses vary in size from a pin's head up to that of a hazel-nut; they may contain well-formed pus, or sanious fluid and *débris*, or more bulky sloughs that have only just been separated. In cases originating in pylephlebitis it may be easy to show that much of the suppuration is in the course of the distribution of the

portal vein. The capsule of the liver is frequently inflamed where abscesses approach the surface.

Symptoms.—Cases of multiple abscesses in the liver are often very obscure, especially when they form a part of a general illness ike pyæmia. There is severe constitutional disturbance, with fever of hectic type, rapid pulse, dry brown or furred tongue, and early prostration. Vomiting is often present, but the action of the bowels is variable; sometimes there is constipation, at others diarrhœa. The liver is mostly enlarged, and in some cases may reach to the level of the umbilicus; it is painful and tender. Jaundice is sometimes, but not necessarily, present; it probably requires the compression by an abscess, or the obstruction by gall-stones, of some larger bile-duct. The condition of the urine and of the fæces as to the bile-pigment will, of course, vary with it. The duration of the illness is from one to several weeks, but the end is certainly fatal.

Diagnosis.—This must depend on the fact that the liver is involved in an acute process, with severe constitutional disturbance, especially if it is associated with some lesion which can be recognised as the primary cause. The presence of jaundice very much facilitates the diagnosis. Where jaundice is absent, it may have to be distinguished from tropical abscess by the uniform enlargement of the liver.

The **Treatment** must be mainly symptomatic. An attempt must be made to improve the general condition by nourishment, quinine, and stimulants. Opium and local applications, poultices fomentations, &c., will be required to relieve pain.

LARGE, SOLITARY ABSCESS

This is met with frequently in India and other tropical countries. It occurs especially between the ages of twenty and forty-five, the intemperate are more liable to it than others, and it has a relation to dysentery, which has been already discussed.

Anatomy.—Tropical abscess is usually, though not always, single, and may reach a considerable size. Its condition varies somewhat with its age: thus a recent abscess presents a ragged inner surface, with little that can be called lining membrane; a somewhat older cyst is lined with opaque yellowish deposit; while very old abscesses are surrounded by a dense fibrous wall formed in the hepatic tissue surrounding them. The pus contained in them is either whitish or yellow, like pus from other sources; or green, and viscid, or curdy; or it may be red or reddish-brown from mixture with blood. It has a peculiar nauseous odour, but it is rarely offensive unless the abscess is near the colon. The abscesses found in the liver in connection with amœbic dysentery always contain amœbæ, and never any so-called pus-organisms. The process is not a true suppuration, but a softening and liquefaction of tissue, already largely necrotic. In abscesses free from

amœbæ, staphylococci (aureus and albus) have sometimes been found. The amount of pus may reach five or six pints or even more.

It is probable that the pus from small abscesses may be absorbed, and in some cases contracted cavities with cretaceous remnants have been found in the liver, indicating a former abscess. But, as a rule, the abscess after a time makes its way to the surface of the liver, where it sets up perihepatitis, and ultimately opens either externally through the abdominal parietes, or into the stomach, duodenum, or colon, or into the abdominal cavity, or by perforating the diaphragm into the pleural cavity, lung, or rarely the pericardium. Sometimes it opens externally through an intercostal space, having first perforated the diaphragm and displaced the lung. It may thus be rapidly fatal, causing peritonitis, pleurisy, or pneumonia; or the cavity may contract after free discharge externally, or into the lung or into the bowel.

Symptoms.—In the early stages these consist of chilliness or actual rigor, followed by febrile reaction. Locally there is pain in the right hypochondrium, which may reach up to the right shoulder; and fulness and tenderness in the same region, with some evidence of enlargement of the organ. There is frequently, but not always, some jaundice. The movement of the ribs in respiration is impeded, and there is a short, dry cough. In the course of a week or a fortnight there may be evidence of suppuration. The liver is enlarged, and may reach far down into the abdomen; the enlargement, however, is not uniform, but there is a marked prominence at one or other part. If there is a large abscess in the right lobe, the ribs of the right side may be elevated so as to cause an obvious bulging of the lower part of the chest, and to increase on that side the costal angle, *i.e.*, the angle between the margin of the ribs and the middle line (*see* p. 670). An abscess projecting from the convex surface of the liver at its back part will cause dulness at the base of the chest by displacement or compression of the lung, diminishing at the same time the breath-sounds, so that an empyema is simulated. If the abscess bulges in front, the surface is smooth, elastic and tense; and fluctuation may be felt, although it is often absent if the abscess is deep-seated and surrounded by a thick layer of hepatic tissue.

The pain is at first dull and heavy, becoming more severe as the abscess reaches the surface, and either sets up peritonitis or stretches the integuments. Pain is felt, also, when the patient lies on the left side, from the falling of the liver towards that side. Both pain and tenderness may, however, be absent. Ascites and marked jaundice are not commonly present; when they are, they result from direct compression of the portal vein or of the large bile-ducts. With the formation of the abscess, the constitutional symptoms become pronounced. The fever is irregular, and of hectic type. Occasionally there are rigors, during which the temperature rises to 103°, 104°, or 105°, falling with profuse

perspiration. The tongue is furred or dry, and vomiting may be frequent, but the bowels are variable.

In course of time there is severe prostration, with emaciation, and the skin assumes the sallow tint common with extensive suppuration. Subsequent symptoms depend on the course which the abscess takes; if it points externally, there is increased prominence at the spot selected, the skin becomes red, tender, and œdematous; its invasion of the chest may cause for some little time the symptoms of compression of the lung above indicated, and finally the chest-wall may bulge opposite one of the intercostal spaces. If it points more centrally, pleurisy, serous or purulent effusion, or pneumonia may occur; or the lung may become adherent to the diaphragm, so that the abscess opens directly into the bronchial tubes, and the purulent contents are gradually expectorated. Rupture of the abscess into the pericardium sets up pericarditis, which is generally quickly fatal. Rupture into the peritoneal cavity may cause a localised peritoneal abscess, or a general suppurative peritonitis, with corresponding symptoms. It is important to remember that sometimes the symptoms of hepatic abscess are entirely latent until one of these accidents by rupture occurs.

The duration of abscess of the liver is variable; it may be from a few weeks to three or more years.

Diagnosis.—The diseases which may be confounded with abscess of the liver are acute perihepatitis, suppurating hydatid (which is, indeed, an abscess of the liver arising in another way), suppurating gall-bladder, subphrenic abscess, empyema, abscess of the abdominal wall and pyelitis. Hepatic abscess, otherwise simulating empyema, often differs from it in the upper line of dulness being convex, and falling near the spine, instead of directly transverse. The fever of hepatic abscess is often mistaken for that of malaria, but will not yield to quinine, as does the latter.

Prognosis.—Some cases get well under treatment, and occasionally this happens when the abscess has discharged into the lung or bowel; but a fatal result is very frequent. On the whole, a small abscess may be considered more favourable than a large one.

Treatment.—In the earlier stages, salines, chloride of ammonium, and ipecacuanha are recommended: the latter should be given in doses of 15 to 20 grains, repeated every six or eight hours. L. Rogers believes that by this drug suppuration can be prevented, and he recognises amœbic hepatitis by a marked leucocytosis, in which the polymorphonuclears are not much increased. When suppuration has occurred, pus should be evacuated, if possible. The abscess should be opened by a free incision, and the cavity should be drained. This may be sometimes done through the anterior abdominal wall; but not infrequently the posterior position of the abscess necessitates operating through the intercostal spaces and the diaphragm. Rogers advocates syphon drainage and irrigation with quinine solution to prevent staphylococcal infection

of the hitherto sterile abscess: and he gives ipecacuanha after operation to prevent the formation of more abscesses. The relief of pain by opium, morphia, and local applications may be necessary.

ACUTE YELLOW ATROPHY

In this remarkable disease the liver undergoes a rapid degeneration of its tissues, and diminishes in size to two-thirds, or even one-half, of its normal bulk.

Ætiology.—It is more common in females than in males, and the majority of patients are under thirty years of age, though it is very rare in children. Indeed, at any age it is a disease of extreme rarity. Its onset is often preceded by severe mental disturbances, and many of the cases have occurred in people who have led a dissipated life, in the subjects of constitutional syphilis, in women of loose habits, and in those who are pregnant. It has also occurred within twenty-four or forty-eight hours of a surgical operation; this has mostly been an abdominal operation, performed under chloroform.

Symptoms.—The symptoms are at first obscure. Often it begins with a jaundice indistinguishable from catarrhal jaundice; or with gastro-intestinal symptoms, such as nausea, vomiting, and irregularity of the bowels; and pains in the hepatic region may occur comparatively early. These symptoms may last two or three weeks, when the more characteristic features develop. These consist of marked cerebral disturbances—at first headache and restlessness, then delirium and gradually developing coma, with convulsive twitchings, or more rarely epileptiform fits, towards the end. Jaundice then appears, or if it has been present early, it becomes deeper. The temperature is rarely high, but may be from 101° to 102°. The pulse, which may have been slow with the early jaundice, now becomes quick. The tongue is dry and brown, and as the symptoms progress, sordes collect about the lips and teeth. There is, besides, pain in the hepatic region, and decided tenderness, which may be recognised even during the stage of coma, if pressure be made there. The extent of dulness diminishes with great rapidity, so that finally its vertical measurement is only an inch or less.

The abdomen is natural, or towards the end it is retracted. The spleen is mostly enlarged. The urine contains both bile-pigment, and bile-salts. But the most remarkable change in the urine is the extraordinary decrease in the amount of urea, uric acid, and salts (chlorides, phosphates, and sulphates), and the presence of two new compounds—leucin and tyrosin. The urea may be entirely absent. Leucin and tyrosin can be obtained by evaporating a small quantity of urine on a glass slide or in a watch-glass, when their crystalline forms may be recognised under the microscope. Leucin forms circular plates, with concentric markings

upon them; tyrosin crystallises in fine needles, which are arranged in sheaf-like bundles or in globular masses. These bodies are products of the cleavage of proteins, and are the substances from which the liver forms urea. They originate in the intestine, and in consequence of the destruction of the liver, are not further transformed, but pass into the urine. The urine not infrequently contains albumin, especially towards the end; and there may be also blood. This appearance of blood in the urine is part of a general hæmorrhagic tendency. There may be coffee-ground vomit; the fæces, which appear to be mostly pale, and deficient in bile, often contain blood; and there may be epistaxis, metrorrhagia, or petechial hæmorrhages under the skin. With increasing coma, death finally takes place, the severer symptoms lasting only from two to four days. Pregnant women, as a rule, abort.

Anatomical Appearances.—The liver is very much diminished in size; it may be only thirty or twenty-eight ounces in weight. It is soft, flaccid, almost like a bag of fluid, and its capsule, which is wrinkled, appears too large for its contents. On section, the liver is of a yellow colour, with patches of rather bright red; or in some parts it is entirely red, in others all yellow. The essential change is a granular and fatty degeneration, by which the hepatic cells are more or less completely destroyed. In the yellow parts of the liver the destruction is less advanced, and some bile-stained cells may still perhaps be found. In the red parts the colour is due to the more complete necrosis of the tissue, by which the vessels are left alone to represent the substance of the liver. Under the microscope, one can often see nothing but granules of albuminous matter, fat and pigment, and larger globules of fat. Leucin and tyrosin are also found in the liver, and will spontaneously crystallise on the surface of sections some hours after death. The bile-ducts are empty, and not stained by bile-pigment; the gall-bladder is also empty, or contains a small quantity of viscid grey mucus.

Other organs undergo fatty degeneration, especially the kidneys, in which the secreting cells are granular and fatty, and the heart and muscles. Petechiæ are found not only under the skin, but in the mucous membranes, under the serous membranes, in the kidneys, and other parts. The blood is thin and fluid, and leucin and tyrosin have been found in it.

Pathology.—The probability of its being an infectious disease is shown by the hæmorrhages, the splenic enlargement, the condition of the blood, and the typhoid, or adynamic condition preceding death; but no specific micro-organisms, other than B. coli communis, streptococci, staphylococci, and pneumococci, have hitherto been found. The jaundice of acute yellow atrophy is possibly explained by acute catarrhal changes in the minute bile-ducts.

Diagnosis.—This generally depends upon the occurrence of cerebral symptoms and rapid diminution of hepatic dulness in a jaundiced patient. Occasionally, however, the jaundice has been

absent. The disease is closely simulated by phosphorus-poisoning, in which the liver undergoes fatty degeneration, and there are jaundice, petechiæ, and cerebral symptoms. But in phosphorus-poisoning the liver is very much enlarged at first if not throughout, and leucin and tyrosin are less abundant in the urine, if present at all. The term *icterus gravis* (Fr. *ictère grave*) used by some writers includes acute yellow atrophy, and phosphorus-poisoning, as well as other fatal cases of jaundice of doubtful pathology.

Prognosis.—Acute yellow atrophy is almost invariably fatal. A few cases are believed to have recovered; in one such case, which occurred at Guy's Hospital, the patient died of the same disease a ew months later.

Treatment.—This must, at present, be purely symptomatic. Sickness, constipation, headache, hepatic pain, and later active delirium or retention of urine, are the conditions that may have to be met.

CIRRHOSIS OF THE LIVER

(*Chronic Interstitial Hepatitis*)

This is a chronic inflammation of the liver, which results in an extensive growth, usually in the course of the portal canals, of a contractile fibrous tissue, whereby the secreting cells of the liver are compressed and destroyed, while the course of the blood through the portal system of veins is seriously obstructed. The liver in advanced cases presents, on section, a number of yellow, brownish-yellow, or brown lobules of hepatic tissue, surrounded and separated from one another by broad tracts of gray translucent fibrous tissue; and it was on account of this generally yellow colour that the term *cirrhosis* (κιρρός, yellow) was used, and not in reference to the presence of excess of fibrous tissue. Nevertheless, the name has been often applied to chronic fibrous changes in other organs of the body—*e.g.*, cirrhosis of the lung and cirrhosis of the kidney.

Ætiology.—In the great majority of cases the cause of cirrhosis is the excessive use of alcohol, in the form of beer, wine or spirits. Little is known as to the amount that is required to produce cirrhosis; there are the widest individual differences. Some people may drink freely all their lives without acquiring it, whereas in others a few months' indulgence seems sufficient for the purpose. In some children that have been the subjects of it, the fact of alcoholism has been proved. But the simple theory of direct irritation by alcohol has not escaped criticism; and the following views have also been advanced: That the real irritant is some toxin produced in the mucus which results from the accompanying gastritis; that the irritant is not alcohol, but some other constituent of the liquid drunk. Where alcoholic excess cannot be proved, a satisfactory explanation is rarely forthcoming. Congenital syphilis is undoubtedly the cause of one variety, and probably contributes to another. It is said that infectious diseases, such as scarlet fever, measles, or

pneumonia, supply the requisite toxin for the occurrence of the common forms of cirrhosis, and much less pronounced degrees of fibrous undergrowth appear to result sometimes from heart disease rickets, very rarely tuberculosis, and perhaps malaria. It is conceivable also that intestinal toxins may cause some forms of the disease.

Cirrhosis of the liver occurs as a late result in some cases of splenic anæmia (*see* Anæmia), and the cases are then described as *Banti's disease*. Rare cases also occur, probably independent of alcohol, of which the subjects are mostly children, and in which there is considerable enlargement of the liver, still greater enlargement of the spleen, stunted growth of the patient, deep pigmentation of the skin, and marked clubbing of the finger-ends (*splenomegalic cirrhosis*). A large-livered cirrhosis is also associated with pigmentation in cases described, some as *hæmochromatosis*, and others as *diabète bronzé*. Of these it is believed that the deposit of pigment from the blood is the first change; cirrhosis results from the irritation of the pigment in the liver; and in bronzed diabetes, glycosuria follows upon its deposition in the pancreas.

The tropical infectious disease, kala-azar, is accompanied by a moderate degree of cirrhosis; a somewhat similar combination of cirrhosis, enlarged spleen, and bone-marrow changes, but without the Leishman-Donovan bodies, is endemic in Egypt (Day and Ferguson); and a curiously localised portal cirrhosis is found as a result of bilharzia infection (*see* Bilharzia hæmatobia).

Anatomical Changes.—The cirrhotic liver varies considerably in size. It may be so large as to reach during life two inches below the level of the umbilicus, and to weigh after death as much as eight or ten pounds; it may be so small as to be inaccessible to touch, and to weigh only twenty-eight or thirty ounces; and it may be of any intermediate size. The larger livers are often smooth, or only slightly granular on the surface; the smaller livers are coarsely granular, or nodular, or present large round bosses, or are distorted into curious shapes. In all cases the organ is very much tougher and harder than normal, from the development of fibrous tissue, which runs in all directions through it. If it can be examined in the earliest stages of cirrhosis, there are found large numbers of leucocytes infiltrating the tissue about the portal canals (Glisson's capsule), and in some cases penetrating more or less between the lobules, or even within them. From these leucocytes is developed white fibrous tissue, which forms a large part of the section in an advanced case. The bands of fibrous tissue running through the organ break it up into islands of hepatic tissue, each of which may consist of several lobules (*multilobular cirrhosis*), or single lobules (*unilobular cirrhosis*); or the fibrous tissue may run between the cells of the lobules (*intercellular cirrhosis*). The cells are atrophied and mostly stained yellow or brown by particles of pigment. In the fibrous tissue are numerous newly formed blood-vessels, which can be injected from the hepatic artery, and, in certain cases, numerous

double rows or cubical cells, which appear to be small bile-ducts. The organ is at first enlarged by the overgrowth of connective tissue, and some large cirrhotic livers also contain a quantity of fat. The fibrous tissue in course of time contracts, and thus compresses more and more the hepatic cells, the branches of the portal vein, and perhaps the bile-ducts. The liver-cells and fat may disappear, and the organ may be reduced much below its normal weight. The varying size of the liver is thus, in part at least, dependent on the stage of the process.

For the most part the difference above mentioned between livers which are deformed, and nodular or bossy, and those which are smooth or only finely granular, corresponds to histological differences, and to clinical differences which will be mentioned later. Thus in the former the fibrous tissue separates groups of lobules: it is a *multilobular cirrhosis*. The organ varies much in size; it may be very large, or about the normal size, or very much smaller: in the last case the shape is often much altered from the extensive and irregular contraction of the fibrous tissue. When the surface is smooth, the fibrous tissue separates single lobules from one another, and it is spoken of as *unilobular cirrhosis*. The organ is often very large—much larger than is common in multilobular cirrhosis; and the shape is little affected. The organ is generally deeply stained with bile, and the fibrous tissue often shows a great number of the double rows of cubical cells above mentioned. Because in the first group of cases the liver is often quite small, and in the second very large, the terms *atrophic cirrhosis* and *hypertrophic cirrhosis* have been employed to distinguish them. But in more than 50 per cent. of cases of so-called atrophic cirrhosis the liver is at the time of death larger than normal, reaching even twice its size; and, on the other hand, the large livers of so-called hypertrophic cirrhosis may certainly contract to the normal size or below it. I have recorded a case in which a liver which reached below the umbilicus, in a patient with strongly marked jaundice and no ascites, was found fifteen months later to have contracted quite close under the edge of the ribs.

An *intercellular* cirrhosis occurs especially in congenital syphilis, the fibrous tissue invading the lobules and running among the hepatic cells.

Symptoms. *Multilobular Cirrhosis.*—The early stage of cirrhosis often passes with very little disturbance. There may be symptoms of congestion of the organ, such as fulness or pain in the hepatic region, with a slight tinge of jaundice; on the other hand, there is frequently, as a result of free drinking, a gastritis which produces loss of appetite, furred tongue, and vomiting, especially in the morning. An examination of the abdomen at this stage may, however, reveal a considerable enlargement of the liver, of which the patient is entirely ignorant. The next symptom is not infrequently *hæmatemesis*, or vomiting of blood; this is due to the commencing obstruction in the portal circulation: as the blood in the portal vein finds a difficulty in passing through the liver, the radicles of this

system, viz., the mesenteric, gastric, and splenic veins, are, of course, congested, and tend to bleed on to the mucous surfaces. But sometimes the blood proceeds from a rupture of the veins at the lower end of the œsophagus, which have become varicose in the course of establishing a free communication between the portal vein and the inferior vena cava or azygos vein. The quantity thrown up is often as much as one or two pints; and occasionally the hæmorrhage is directly fatal. The vomiting may be followed or accompanied by melæna. Piles are not infrequently present at the same time; and hæmorrhage from other parts (gums, nose, and lungs) is liable to occur in the course of cirrhosis.

The most important and constant result of the portal obstruction is the effusion of fluid from the distended veins into the peritoneal cavity, constituting the form of dropsy already described as *ascites* (*see* p. 768). In many cases, when ascites has developed, the liver is still enlarged, and can be felt one or more inches below the ribs; if the fluid be displaced by the hand (*see* p. 769), the surface is felt to be rough, granular, or nodular, and the edge is rounded. If the organ has already contracted below the normal, it may be impossible to feel it; but the results of percussing the lower ribs to estimate the vertical extent of hepatic dulness are always uncertain in such a case, since the presence of fluid allows considerable change in the relations of the liver, intestine, and abdominal walls to one another. The spleen is often enlarged and may be felt; it is frequently from 20 to 30 ounces in weight. The peritoneum is often thickened and opaque, both in its visceral and parietal layers. The surface of the abdomen is covered by large veins, running between the iliac and thoracic trunks. This collateral circulation is partly, perhaps, due to the compression of the vena cava inferior by the fluid in the abdomen, for the feet and legs are often œdematous at the same time; but it is also a means by which the portal circulation is relieved. This is an important point, for it must be remembered that the portal system is not completely shut off from the general circulation, but that there are, even in health, means of communication which in cirrhosis become greatly enlarged, and allow of some of the blood in the portal vein radicles reaching the right side of the heart without passing through the liver itself. Those which have been described are communications (1) between the gastric and œsophageal veins at the opening in the diaphragm; (2) between the inferior mesenteric and the hæmorrhoidal branches of the internal iliac vein; (3) between the coronary veins of the stomach and branches of the phrenic veins; (4) between branches of the mesenteric vein and the spermatic vein, or others in the abdominal wall. Frerichs described (5) vessels forming in the adhesions between the liver and the diaphragm; and (6) a large vein (accessory portal of Sappey) has sometimes been found running along the round ligament of the liver, by which the portal vein communicates directly with branches of the epigastric and internal mammary. Sometimes a loud venous murmur is heard at the epigastrium.

The bases of the lungs are often seriously compressed by a large ascites, and the heart is displaced upwards. The urine is generally scanty, high-coloured, with abundant deposits of red urates, and not infrequently a trace of albumin. The last may be due to pressure on the renal veins; it is unsafe to diagnose co-existing granular kidneys from that fact alone.

By the time that ascites is well developed, the patient is in other respects often seriously ill. He is thin, weak, with sunken eyes, a slight tinge of jaundice, and small stellate venules on the face. The temperature is mostly normal, but fever is sometimes present.

The symptoms remain much the same, but the prognosis is very unfavourable. Sometimes recovery follows the use of diuretics and purgatives, and the removal of the fluid by tapping, and is no doubt largely due to the development of the venous anastomosis above noted. But death often results within a few months of the appearance of ascites, with cardiac failure, or with cerebral symptoms (delirium and coma), which may ensue at the very time that the fluid is becoming absorbed, and may carry off the patient in a few days. This mode of death is probably due to a toxæmia resulting from the imperfect performance of the hepatic functions. Occasionally hæmatemesis, or hæmorrhage from the gums or nose, or peritonitis after tapping is fatal.

Unilobular Cirrhosis.—In the cases for which this name has been reserved the earlier indications that alcoholic excess is injuring the liver are not confirmed by ascites, but the main symptom is a more or less intense jaundice. The liver is found to be very large indeed, reaching one or two inches below the umbilicus, giving a certain degree of prominence to the abdomen, and an area of dulness which may measure vertically nine or ten inches. The surface may be smooth or distinctly granular to the touch. The urine is stained with bile-pigment, but the fæces generally retain their natural colour; and the jaundice is attributed to the obstruction of some of the small ducts within the liver. After a time the patient is taken with all the symptoms of acute blood-poisoning; he becomes delirious, even violently so, and relapses into coma. The temperature is high, hæmorrhages occur under the skin and from the mucous membranes, and he dies in three or four days. But ascites may occur before the fatal symptoms: and, as already mentioned, contraction may take place, and probably ascites supervenes upon this.

Diagnosis.—Cirrhosis is often latent until hæmatemesis, ascites, or marked jaundice discloses the secret; it has been already stated that examination may discover an enlarged rough liver in a tippler who has no decided trouble. Most commonly the diagnosis has to be made when ascites has already appeared, and then the history of drinking and of hæmatemesis, the presence of an enlarged liver, enlarged spleen, and slight jaundice, are sufficient to determine the case. Of the other conditions of the liver and peritoneum causing ascites, the most important are *cancer*, which may obstruct the portal

vein, or its largest branches, and the association of *perihepatitis* with *chronic thickening of the peritoneum* (*see* Perihepatitis). *Cancer* and *tubercle*, apart from the liver, also cause a peritonitis, which results in ascites. The former may be recognised by the occurrence of nodules of growth in different parts of the abdomen. The latter often presents a thickening of the omentum, which may be mistaken for an enlarged liver. But the resemblance between atrophic cirrhosis with ascites, and tubercular peritonitis with liquid effusion alone, may be very close: and the diagnosis may be possible only after a tapping, when the abdominal organs can be better felt, and the fluid can be examined bacteriologically; or if there is evidence of tubercle in other parts of the body. Hæmatemesis is more frequently the result of cirrhosis than of any other disease except gastric ulcer or erosion, and is valuable in diagnosis.

When the chief symptom is jaundice, and there is no ascites, the liver is mostly enlarged, and nearly smooth on the surface. An infiltrating cancer of the liver may closely resemble a large-livered cirrhosis, and the consistence of the organ may be very similar in the two cases. The persistence of bile-pigment in the stools shows that the larger ducts are not obstructed, and rather favours cirrhosis.

The not infrequent coincidence of multiple neuritis and cirrhosis from alcoholism should be borne in mind.

Prognosis.—This is very bad. When ascites appears, the future course is often only a few months; on the other hand, repeated tappings with complete change of habits may prolong life for years. Large-livered cirrhosis without ascites may last three or four years.

Treatment.—Little, if anything, can be done with the cirrhotic liver itself; and treatment resolves itself into the prevention of further mischief, and the attempt to obviate the effects of the damage already done. In alcoholic cirrhosis the first essential is that the ingestion of alcohol should be absolutely stopped: and in early stages, where the liver is still uncontracted, and ascites has not yet appeared, the liver may regain its normal size, and the patient his health. It is, however, impossible in such a case to say how far fibrosis had progressed. The diet should be light and easily digestible; the bowels should be kept active, and sickness and any dyspeptic symptoms may be treated by effervescing salines, bismuth and bitter tonics. When ascites occurs an attempt to promote its absorption must be made by the use of diuretics and purgatives. Of the former, acetate, nitrate, and bitartrate of potassium, spirits of nitrous ether, squills, and digitalis are most frequently given; and the resin of copaiba in doses of 15 grains three times a day sometimes has a good effect. But the kidneys act at a disadvantage from the pressure of the ascitic fluid. Of purgatives, sulphate of magnesium, bitartrate of potassium, compound jalap powder, or elaterium may be employed. If these fail to remove the fluid, and the abdomen becomes very tense, paracentesis is required, and it may sometimes be repeated with success as the fluid reaccumulates. Attempts to develop a collateral circulation, based on the view that

ascites is mainly mechanical in origin (*see*, however, p. 768), have been made (1) by opening the abdomen, scraping the peritoneum on the opposed surfaces of the liver and diaphragm, and bringing them into contact by stitches (Drummond and Morison, Talma); and (2) by uniting the great omentum to the anterior abdominal wall *epiplopexy*).

These operations have not been highly successful.

OTHER FORMS OF ATROPHY AND CONTRACTION OF THE LIVER

Some atrophy occurs as the result of old age or of inanition. The organ may be reduced to half its normal bulk, but there is no alteration in its structure and no induration, the lobules being diminished in proportion to the size of the whole organ. It produces no symptoms. Partial atrophy may result from the pressure of adjacent organs or of tight-lacing: the right lobe of the liver is elongated downwards, and where it is compressed between the lower ribs and the right kidney, the hepatic tissue is atrophied, and replaced by fibrous tissue in a transverse line below which a portion of liver extends down to the umbilicus level.

Perihepatitis also causes a certain amount of atrophy

FATTY LIVER

The hepatic cells normally contain a small quantity of fat in the form of minute globules. Under certain conditions of disease the fat is immensely increased, and each cell may contain such a large amount that the nucleus and outline of the cell are entirely obscured, and the cell itself might be supposed to be destroyed. This change takes place first at the periphery of the lobule; later the whole is invaded. The liver is much enlarged, it has a smooth surface, is somewhat rounded at the edge, on section has a whitish-yellow colour and uniform appearance, and it may actually float in water. This *fatty infiltration* occurs under two sets of conditions. One is in association with general obesity and fatty degeneration of other organs, such as the heart and muscles. A similar change takes place in some drinkers. The other follows emaciating disease, especially phthisis, and sometimes cancer. In phthisis it may be explained by supposing that the fat cannot be disposed of from defective respiratory powers.

The fatty liver is painless; it can readily be felt as a large, smooth organ in phthisis; but it may be less easy in obesity, on account of the thickness of the abdominal walls. Dyspeptic symptoms and deficient secretion of bile are referred to fatty liver.

A partial form of fatty change occurs as a result of long-continued congestion in the nutmeg liver of heart disease; the fatty infiltration is most marked at the periphery of the lobules.

As contrasted with the above deposit of fat in hepatic cells, which still persist intact, an actual *fatty degeneration* with complete destruction of the cell takes place as a result of acute yellow atrophy, and notably in poisoning by phosphorus.

LARDACEOUS DISEASE OF THE LIVER

Lardaceous degeneration has been already referred to in connection with empyema and phthisis; and as the liver is one of the organs which is most frequently implicated, a short account of the degeneration must here be given. It consists in the deposition in the tissues of a firm, colourless, translucent material (*lardacein*), which is stained by certain colouring-agents. Thus, iodine in tincture or aqueous solution turns it a rich brown-red or claret colour. The iodine may be applied to a section of the fresh organ, after this has been washed free of blood, and the affected parts are then mapped out by the characteristic tint. The subsequent addition of dilute sulphuric acid changes this to a dark purple hue. Methyl-violet or gentian-violet turns lardaceous matter red, while the surrounding healthy tissue is stained blue.

Lardacein, which was at first thought from the iodine reactions to be of a starchy nature, is a combination of chondroitin-sulphuric acid and proteid. The old terms, *amyloid substance*, and *amyloid liver*, are therefore incorrect. Lardacein is very resistant to chemical action and to putrefaction.

The tissues in which it is found are, first in point of time, the walls of the blood-vessels; secondly, various connective tissues; and lastly, if at all, the gland-cells of an organ. Indeed, the material is mostly intercellular in its position: thus it is found in the small arteries deposited between, and separating from one another, the muscle fibre-cells of the middle coat; in the spleen it exists as streaks and patches between the cells of the pulp; and in the liver it lies in similar particles between the capillaries and the gland-cells. It is, indeed, not so much a degeneration as an addition to the structure; and solid organs affected by it are generally much enlarged. Its relation to the vessels suggests that it is deposited from the blood.

It occurs most often in the spleen, kidneys, liver, intestines, and stomach; and with decreasing frequency in the suprarenal capsules, lymphatic glands, thyroid, aorta, ovaries, and uterus.

The lardaceous deposit can, in the vast majority of cases, be attributed either to prolonged suppuration from phthisis, syphilis, tubercular disease of bones and joints, and empyema, or to syphilis, without suppuration. Other cachectic conditions have been present in a few instances. The way in which these causes operate is still uncertain, but it appears likely that bacterial toxins may be the intermediate agent.

In the liver the lardaceous change is first observed in the middle

zone of the lobules, where the capillaries are most intimately connected with the divisions of the hepatic artery. As the deposit increases the hepatic cells are compressed and atrophied, but they are only occasionally the seat of lardaceous deposit. The liver becomes enormously enlarged, has a smooth surface, and somewhat rounded edge, and is entirely free from pain or tenderness. The disease causes no jaundice. It is accompanied by the signs of the causative disease, and often by an enlarged spleen, albuminuria, and diarrhœa, the results of the deposit in other organs. A lardaceous liver, which is at the same time the seat of syphilitic gummata or cicatrices, naturally loses its uniform smooth surface, but may be recognised by its other associations. The portal circulation is not obstructed by the lardaceous change, and although ascites is not infrequently present, it is mostly associated with general anasarca, and must be referred with it to co-existing disease of the kidneys.

Prognosis.—This is very bad, but decrease of the enlargement after efficient surgical treatment has been recorded.

Treatment.—The cause must be, if possible, removed. This is impracticable in phthisis; but other sources of suppuration may perhaps be treated surgically; and potassium iodide, cod-liver oil, iron, quinine, and other tonics should be given. Mercury and potassium iodide should be used in syphilitic cases

SYPHILITIC DISEASE OF THE LIVER

This may be *congenital* or *acquired*.

Congenital syphilis occurs in two forms: first, as an interstitial hepatitis; secondly, as gumma. The diffuse change begins as a cellular infiltration, which develops into a fibroid induration, and is generally most marked in the interlobular spaces; but sometimes it invades the lobules, and surrounds each cell with a layer of fibrous tissue, constituting a true intercellular cirrhosis, and leading to considerable enlargement of the organ. The spleen is often enlarged at the same time: jaundice occasionally occurs, but ascites rarely. Gummata and cicatrices are less common than in the acquired disease. A multilobular cirrhosis has sometimes developed in those previously the subject of the intercellular form.

Treatment.—Hydrargyrum cum cretâ should be given, or mercurial inunction should be employed for some months, and their use should be intermittently continued for two or three years.

Acquired syphilis also produces diffuse hepatitis and gummata, but the latter are much more frequent. They present the general features of gumma in other situations, being more or less spherical yellow masses, tough and elastic, surrounded by a zone of gray fibrous tissue, from which branch off numerous bands into the adjacent hepatic substance. The contraction of the fibrous tissue produces a depression or fissure on the surface of the liver, at the

bottom of which lies the gumma which has caused it; and so the organ may become coarsely lobulated and deformed. Gummata not infrequently break down in the centre into a puriform detritus; on the other hand, they may become completely fibrous, so that nothing remains but a depressed scar; or calcareous granules may be deposited in them. Livers affected with gummata often undergo the lardaceous degeneration, in consequence of which they may be of large size in spite of cicatricial contractions. Perihepatitis is another change resulting from syphilis.

Symptoms.—Occasionally a large gumma may form a prominence on the anterior surface of the liver, smooth and elastic, and strongly suggestive of a hydatid or other cyst. Like other localised enlargements it may cause elevation of the right costal margin, and enlargement of the right costal angle (*see* p. 670). More often, but probably in later stages, syphilitic livers are large, hard, irregular on the surface, and deformed, from the contraction of the fibrous cicatrices. Neither ascites nor jaundice is necessarily present, but in particular cases they may occur from the pressure of a gumma upon the portal vein or the bile-duct; and there is often albuminuria from co-existing lardaceous disease of the kidney. A gumma is sometimes accompanied by decided fever of hectic type.

Treatment.—In early cases, iodide of potassium will quickly reduce the gumma and check the fever accompanying it; but when there are old cicatrices and extensive lardaceous disease, little good can result, beyond, perhaps, the prevention of further mischief.

TUBERCLE OF THE LIVER

This is almost invariably a part of a general tuberculosis. It occurs either as minute grayish-yellow granulations, less than a pin's head in size; or of somewhat larger (3 to 5 mm.), bright yellow masses, more easily detached from the surrounding liver-tissue. These larger masses are often softened into a cavity in the centre, which is deeply stained with bile. As a rule, no local symptoms accompany hepatic tuberculosis; but occasionally a general enlargement of the liver results; and in rare cases there is jaundice (*see* Miliary Tuberculosis, p. 154).

NEW GROWTHS IN THE LIVER

The only tumour of the liver that is at all common is carcinoma. Of others, cavernous angeiomata, simple cysts, and the lymphomatous deposits associated with Hodgkin's disease are the most frequent. They rarely cause definite symptoms. Cases of spindle-cell sarcoma, melano-sarcoma, cysto-sarcoma, myxoma, and adenoma have been recorded.

CARCINOMA OF THE LIVER

Pathology.—Primary carcinoma occurs, according to Ziegler, in three forms—as nodules appearing in any part of the liver, as a diffuse infiltration, and as a growth confined to the interlobular connective tissue. In its structure it often consists of a typical clump of epithelial cells; or they may be formed on a glandular type with cylindrical cells. But more than three-fourths of the cases of cancer of the liver are secondary to cancerous deposits, either in the liver itself or in other organs, especially the stomach and pylorus, the small or large intestine, the gall-bladder, the glands in the portal fissure, the uterus, the female breast, or the vertebræ. A not infrequent antecedent of cancer is the presence of gall-stones in the gall-bladder or in the cystic or common duct. The cancer-cells are carried to the liver by branches of the portal vein, and lodged in the lobular capillaries. The form of the secondary cancer, whether soft or hard or melanotic, is determined to a certain extent by the nature of the primary growth.

If the cancer is diffused, the liver is merely enlarged; but when it exists in the form of nodules, or separate tumours, the liver takes at the same time the most varied shapes. Each nodule tends to grow evenly in every direction, and thus to keep a globular form, and when it reaches the surface it will project as a hard, convex, or hemispherical outgrowth. But as the nodules become larger—for instance, one and a half to two inches in diameter—they often break down in the centre into granular and fatty detritus, and as a consequence those that project on the surface, being unsupported on one side, sink in and form a central depression or *umbilication*, a condition which may sometimes be felt through the anterior abdominal wall. The lower edge of the liver is also irregular and nodulated. On section, such a liver presents irregular areas of white cancer growth, with a more or less circular outline; the larger ones are softening in the centre, and many of them are blotched by hæmorrhages. The intervening hepatic tissue is often of a deep brown or yellow colour. Where the cancer has started from the gall-bladder, or the bile-duct, or has grown in from the portal fissure, the growth is most extensive in that region, or may be quite confined to it. Sometimes the empty gall-bladder, or a gall-bladder containing some calculi, is embedded in a mass of cancer. Cancer nodules near the portal fissure may compress the bile-duct or the portal vein, and the latter may be entirely filled by the new growth.

Ætiology.—The chief features in the ætiology of cancer of the liver are its relations to the various primary lesions above mentioned, and to gall-stone troubles of older date. Like cancer elsewhere, it is most common between the ages of forty and sixty.

Symptoms.—Cancer of the liver usually gives rise to a good deal of pain, affecting the right hypochondrium, shoulder, and

loins. At first not much more than a sense of weight and uneasiness, it afterwards becomes severe and lancinating, and is accompanied by tenderness. Occasionally, however, pain is absent. The liver, as already stated, is enlarged; it may reach far below the umbilicus, and over towards the left side; the nodules are prominent on the surface, and the irregular outline may even be seen in profile. For the most part the enlargement is in a downward direction, but large masses may grow from the convex surface, and force up the diaphragm so as to compress the base of the lung. The surface of the cancerous mass is, as a rule, of almost stony hardness, distinctly more hard than cirrhosis, or lardaceous disease, and the transition from hard cancer to the soft normal tissue can often be recognised. Jaundice occurs in about half the cases, and can generally be shown to result from pressure on the main bile-duct, especially in those cases where the cancer starts from the portal fissure. Similarly, ascites is often, but not always, present, and rarely is the fluid as abundant as it may be in cirrhosis. It mostly depends on direct pressure on the portal vein or its large branches; occasionally on a co-existing peritonitis. The emaciation, pallor, and prostration common to malignant diseases of the abdominal viscera are also present. Pyrexia occurs in many cases of cancer of the liver; and occasionally it has exacerbations and remissions like those seen in Hodgkin's disease (*see* p. 864).

Diagnosis.—A jaundice of some months' standing in an old person with an enlarged liver is, in the majority of cases, due to cancer of the liver or of the head of the pancreas; though occasionally the bile-duct may be obstructed permanently by a gall-stone. If nodules of cancer can be felt on the liver, the diagnosis of hepatic cancer is certain; if the liver is of uniform and not very great hardness, cancer is only probable. In cases without jaundice, the large, irregular, and bossy liver, and the emaciation of the patient are generally distinctive. Lardaceous and cirrhotic livers are less hard and more uniform. In both these cases the spleen is frequently enlarged also: in the first case by lardaceous deposit, in the second by venous stagnation; whereas cancerous enlargement of the spleen is relatively uncommon. Syphilitic livers may be irregular and painful, but often occur in younger people, and have their own special history. A long history of gall-stones does not exclude, but rather favours, the possibility of cancer.

Prognosis.—This is hopelessly bad. The duration is rarely more than twelve months, but may be two or three years. The softer forms of growth may kill within a month or two.

Treatment.—This can be only palliative, and consists in relieving pain and in meeting other symptoms, mostly of the digestive organs, such as vomiting, flatulence, and constipation. The diet should be light but nutritious, and, considering the functions of the liver, saccharine and oily substances should be sparingly given.

HYDATID OF THE LIVER

Hydatid tumours are cysts which contain a colourless, non-albuminous liquid, and which arise as a stage in the development of an intestinal worm, the *Tænia echinococcus*. They may occur in any part of the body, such as the brain, lung, spleen, peritoneal cavity, intermuscular spaces, or spinal canal, but are most frequent in the liver; and accordingly the description of their growth will be given in this place.

The *Tænia echinococcus* is a minute tapeworm, measuring only one-quarter of an inch and consisting of four segments, of which the first has hooklets and suckers, and the last, longer than the other three put together, forms the mature *proglottis* (*see* p. 755). This worm inhabits the intestines of the dog, and its cystic form infests the sheep, just as the cystic forms of the human *Tæniæ* are found in pigs and cattle. If the ova of the dog's tænia by any accident reach the human intestine, an embryo in due time escapes, and finds its way to the liver, where it loses its hooks, and is transformed into a vesicle or cyst, containing a clear liquid. The wall of the cyst consists of an outer laminated very elastic layer, and an inner *parenchymatous* layer, containing granular matter, cells, muscle-fibres, and a vascular system. As it grows it sets up a certain amount of irritation in the tissue around, and a layer of fibrous tissue is developed in immediate contact with it. The liquid is clear, or just opalescent, of specific gravity 1005 or 1007, and free from albumin; but it is said to contain a small quantity of glucose and succinate of ammonium.

As the cyst grows it may be reproductive in three ways. First, when the cyst has reached the size of a walnut, it develops from its inner parenchymatous layer smaller cysts, which remain attached by a pedicle, and in which are formed from three to six or more *scolices* —that is, small cyst-like bodies with four suckers and a ring of hooklets at one end. The cysts containing them are called *brood-capsules*, and are sometimes so numerous as to give a velvety appearance to the inner surface of the hydatid cyst. Secondly, the original cyst produces so-called *daughter-cysts*, either directly from the brood-capsules which then become detached, or by independent growth between the two layers of the cyst and subsequent discharge into its interior. The daughter-cysts have the same structure as the original, or *mother-cyst*, and may produce within themselves *grand-daughter-cysts*. Thus, a mother-cyst may contain hundreds or thousands of cysts, of all sizes, from a pea upwards. This is called *endogenous* cyst-formation. Thirdly, daughter-cysts formed between the layers may be discharged externally into the liver (or other organ), constituting *exogenous* cyst-formation; and this is more common in animals. A hydatid cyst may be sterile, producing neither brood-capsules nor daughter-cysts.

A very rare kind of hydatid is the *multilocular hydatid*. It forms

a hard globular mass, consisting of a number of cavities or alveoli, about the size of peas, with transparent, jelly-like contents, which Virchow showed to be the remains of hydatid cysts. Scolices are sometimes found, but the cysts are mostly sterile.

The ordinary hydatid cyst may grow to an enormous size, so as to contain several pints of fluid, and it thus constitutes a tumour which exerts considerable pressure on surrounding parts.

Changes in the Cyst.—A hydatid cyst may last for several years, without any essential change beyond its growth; but its existence may be shortened (1) by spontaneous rupture; (2) by death and conversion into a harmless mass; and (3) by suppuration. It is not quite clear what is the exact cause of death, whether the entrance of bile into the cyst, or the impaired nutrition of the daughter-cysts, due to the rigidity of the capsule. In any case, the result is that the hydatid is converted into a mass of opaque membranes, more or less closely packed together, and mixed with a yellow pasty or putty-like material, in which calcareous salts and cholesterin crystals can be recognised. Very rarely the contents of the cyst are converted in a gelatinous substance, containing abundance of albumin and chlorides (Bruce and Sheild). If suppuration takes place the hydatid is also killed, and the abscess thus formed contains shreds of hydatid membrane, and the silicious hooklets from the heads of the scolices. On the other hand, the spontaneous rupture of a cyst is followed by suppuration.

Symptoms.—Of these the most important is the swelling which the liver, enlarged by the presence of the hydatid, forms in the upper part of the abdomen. If the cyst is deeply seated, or on the upper surface of the liver, the swelling may be entirely due to the displaced or enlarged liver; if the hydatid is near the anterior surface, it forms a distinct globular or hemispherical prominence, which is tense, elastic, and if of sufficient size, distinctly fluctuating. Such cysts sometimes present what is known as the *hydatid thrill*. If the finger or fingers of one hand are laid on the tumour, and are struck with the tips of the fingers of the other hand, a vibration is set up which can be felt for some little time by those of the hand still applied. It is, however, by no means always present, and it is of doubtful significance, since it is probably due merely to the tension of the cyst-wall, and may therefore presumably be obtained in other cysts, if tightly filled.

Cysts which do not merely project from the lower surface of the liver, but occupy its substance or upper part, frequently exert a local pressure upon the ribs, diaphragm, and right lung, with the same results as are seen in hepatic abscess, viz., bulging of the right side of the chest, elevation of the lower ribs, enlargement of the right costal angle, and dulness of the right base, with diminished breath-sounds and diminished tactile vibration (*see* p. 774).

A comparatively small cyst may happen to press on the portal vein and cause ascites, or on the bile-duct and cause jaundice.

Pain is not generally a prominent symptom in hydatid tumour of

the liver, and it may be entirely absent. Or it may be severe, even when the tumour is small. It sometimes depends on the occurrence of peritonitis over the cyst, or on the size of the cyst being such as to cause much local tension. The health of the patient is generally good, and is at first entirely unaffected by the presence of the hydatid; but sometimes there are attacks of urticaria, and in most cases there is a moderate excess of eosinophile leucocytes in the blood, reaching 8 per cent. or more.

Suppuration of the cyst is commonly indicated by the onset of pain, or by its increase if formerly present; the patient loses health and strength, and has elevation of temperature, and perhaps rigors. He has, indeed, an abscess of the liver, with its accompanying conditions; and this abscess may, similarly, point and discharge its contents in different directions. Rupture through the abdominal parietes has occurred with varying results; perforation into the stomach or alimentary canal may be followed by recovery, daughter-cysts and portions of the mother-cyst escaping by the fæces or by vomiting. Or the cyst opens into the base of the lung, and pus, cysts, and bile-pigment are expectorated, also with a favourable result in some cases. Rupture into the pleura, pericardium, or peritoneum is nearly always fatal, the exception being most likely to occur in the case of the peritoneum. Sometimes the hepatic vein or the inferior vena cava has been opened, and daughter-cysts have been carried into the right ventricle and have blocked the branches of the pulmonary artery; and the portal vein has also been invaded. If the cyst communicates with the biliary passages, bile stains the daughter-cysts, and causes the death of the parasite. Conversely, the cysts may lodge in the bile-ducts and cause jaundice.

Diagnosis.—Hydatid is distinguished by its being a localised swelling of the liver, of long duration, and not at first affecting the health of the patient. Where it is accessible to palpation, its round, elastic, and fluctuating properties distinguish it from most other enlargements of the liver. Sometimes a hydronephrosis may closely simulate a hydatid; but generally it is distinguished by a lower position, by the colon lying in front of it, and by the slightness of its movement on inspiration. The test of a hydatid cyst is the nature of the fluid drawn from it by aspiration or trocar and cannula. This has the properties already described; in addition, it may contain some flocculi, consisting of detached scolices, or the minute hooklets which are their most characteristic features. These measure about 25μ in length, are slightly curved, and present a prominence on the concave side which makes them almost triangular.

When suppuration has taken place, it is indicated by the local pain, tenderness, and prominence, combined with the constitutional disturbance; and the previous history will generally help to distinguish the hydatid from the tropical or pyæmic abscess.

In the case of a supposed pleuritic effusion, hydatid may be suspected if there is disproportionate displacement of the liver

downwards, or if there are other hepatic symptoms. It is proved by the withdrawal of clear hydatid fluid, or of thick fluid stained with bile, or of thick grumous or offensive pus containing hooklets.

The method of serum-diagnosis employed in syphilis (*see* p. 143) is available in the case of hydatid cysts. The serum of patients with hydatid disease has been found to contain specific anti-bodies, which in combination with antigen contained in hydatid fluid, will fix the complement, and so prevent hæmolysis. The reaction is not prevented by the suppuration of the cyst, nor by the entry of bile into it.

Treatment.—Drugs have no influence upon the growth of the parasite. The fluid may be withdrawn from the cyst by the *aspirator*, and this of itself appears to be fatal to the parasite; at any rate, a cure has often followed. In a few cases success has attended the method of *electrolysis*, by means of needles inserted into the cyst and connected with a galvanic battery.

But the best treatment is to make a free *incision*, and remove the cyst completely. Adhesion of the liver to the parietes may be secured before the cyst is opened; a drainage-tube is kept in, and the liver gradually contracts. If the cyst has already suppurated, it must be opened and treated in the same way as the tropical abscess.

Prevention.—Since the hydatid of man is obtained from the tænia echinococcus of the dog, and that is propagated by means of the sheep and pig, it is desirable—first, to prevent dogs from eating offal from sheep and pigs; and secondly, to destroy the tapeworms (or their ova) developed in the dog. For this last purpose it has been recommended to purge dogs periodically, and to burn or bury their excreta; further, to scald frequently the floors of their kennels.

CONGENITAL FAMILY CHOLÆMIA

(*Congenital Acholuric Jaundice*)

This is a form of jaundice, to which several names have been given, representing its occurrence in early life, and in many members of the same family, the absence of bile from the urine, and the presence of an enlarged spleen. The jaundice is often noticed immediately after birth, or develops slowly at a later time: it may persist for years, or it clears up and recurs from time to time. It is not an obstructive jaundice, for the fæces retain their normal colour, and the urine is generally, except in aggravated attacks, free from bile-pigment; but it contains urobilin. The blood, on the other hand, contains bile-pigment, but is free from urobilin, or urobilinogen. The blood shows other important changes indicative of anæmia: thus the red corpuscles are reduced to 3,000,000 or 3,500,000, and present moderate degrees of poikilocytosis, anisocytosis, polychromasia, and punctate basophilia; while

nucleated red cells are present. The hæmoglobin is reduced to 50 or 45 per cent., and the colour-index is slightly below unity. The leucocytes are generally fewer than normal, but occasionally there is leucocytosis; the large lymphocytes are sometimes in excess, and a few myelocytes may be present. The spleen is enlarged, and appears to grow harder with the progress of the case. The liver is only slightly enlarged, and has not been known to be cirrhosed; it often enlarges during exacerbations and diminishes afterwards.

A striking feature in the blood is the fragility of the red corpuscles, which are broken up (*hæmolysis*) by sodium chloride solution at much lower degrees of dilution than will affect normal corpuscles.

The anæmia, the alterations in the blood, the persistently enlarged spleen, and the absence of bilirubin from the urine, render it extremely probable that the primary change is in the blood-forming organs, and that the jaundice is a *hæmolytic* jaundice: and the spleen may be enlarged in consequence of the excessive hæmolysis.

The patients, as children, are not generally stunted as in splenomegalic cirrhosis (*see* p. 779): nor are the fingers clubbed. Moreover, they may be little troubled by their complaint, and may live for many years.

The only feature in the ætiology is its occurrence in several members of one family: and congenital syphilis does not appear to be a cause. Arsenic appears to have no influence upon the disease

CATARRHAL JAUNDICE

(*Catarrh of the Bile-Ducts; Catarrhal Cholangitis*)

This is one of the commonest forms in which jaundice occurs. It has been generally believed to be caused by obstruction of the common bile-duct by catarrhal inflammation, which has spread up from the duodenum, so as to cause thickening of the mucous membrane of the duct, with or without some excess of its secretion. As the patient almost invariably recovers, opportunities of verifying the diagnosis are quite exceptional; and it is possible that some cases called catarrhal jaundice may really be due to compression of the duct by acute or subacute inflammation of the head of the pancreas.

Ætiology.—Catarrhal jaundice is especially frequent in early life. It is often associated with evidences of gastro-duodenal catarrh, but these are not always present. It may also be set up by local diseases of the bile-ducts, such as gall-stones, but here the jaundice results from the gall-stone, and any symptoms due to the secondary catarrh would be subsidiary. It is usual to associate with catarrhal jaundice the well-known instances of jaundice from fright, the main features of which are, at any rate, similar; and also the cases of "epidemic jaundice," in which it seems likely

hat a toxic agent is at work. The jaundice of infectious diseases, pneumonia, typhoid, syphilis, &c., may possibly be catarrhal.

Symptoms.—The patient may have indigestion, weight, pain, or distension of the stomach after food, with, perhaps, occasional sickness for three or four weeks before the jaundice; and in other cases it may occur after unusual indulgence in particular kinds of food; but in very numerous instances the patient knows absolutely nothing of his illness until he himself sees in the looking-glass, or is told by his friends, that his skin is acquiring a yellow tinge. Occasionally, the jaundice is preceded by severe pains in the limbs. In catarrhal jaundice the skin and conjunctivæ are of a bright yellow colour, the urine is yellowish-brown, or as dark as porter, and gives the play of colours with nitric acid; the fæces are pale or clay-coloured. The temperature is generally normal, and there may be no constitutional disturbance, the patient being able to do his work as usual; but often he is languid, indisposed for exertion, with a bad appetite, and some nausea. There is mostly no pain in the hepatic region, and not even tenderness; but both may be present in moderate degree. The liver, also, is often not at all enlarged, but sometimes its dulness reaches one or two finger-breadths below the margin of the ribs, and the edge may then be felt, as well as the distended gall-bladder. The bowels are variable, most often constipated, occasionally loose. The pulse may be unaffected, but it is especially in this form of jaundice that abnormally slow pulses have been recorded.

The illness lasts from two to five or six or more weeks, and the jaundice gradually disappears, the urine becoming normal in colour first, and the skin more slowly recovering.

The **Diagnosis** is generally easy. The painless, or almost painless, onset of jaundice in a young person, previously healthy, and presenting no considerable enlargement of the liver, as a rule, distinguishes it from the jaundice of *gall-stones*, of *cancer*, and of *cirrhosis*, the other most common causes. *Acute yellow atrophy* may begin with a jaundice, which is in no respects different from catarrhal jaundice, and which lasts from three to five weeks before the onset of the serious symptoms. But the disease is extremely rare, and there are never any signs by which one can anticipate its occurrence in a given case.

Prognosis.—With the above exception it is entirely favourable.

Treatment.—The patient need not be confined to bed, nor even to the house, but should take a light, simple diet, and avoid stimulants; and a saline laxative should be given if the bowels are actually confined. Sometimes the jaundice passes off quickly with little else, but in many cases recovery appears to be hastened by the internal use of alkaline remedies, especially the bicarbonate of sodium in combination with rhubarb, taraxacum, or calumba. Sodium salicylate is also said to be useful. Carlsbad and Vichy waters are often recommended, the former especially for its laxative action. German writers advise daily injections *per rectum* of one or two

quarts of water at a temperature of 60° to 90° F., to be retained as long as possible. It is said to be proved that the injections cause contractions of the gall-bladder, which may overcome the mucous obstruction in the common duct. Compression and faradisation of the gall-bladder in cases where this is palpable have also been employed; but the former is not entirely free from risk.

SUPPURATIVE CHOLANGITIS

This is always due to infection by micro-organisms, *e.g.*, streptococci, staphylococci, pneumococci, the typhoid bacillus, and B. coli communis; and is either determined by local diseases, such as gall-stones, cancer or hydatid diseases, or by the more general infections of influenza, pneumonia, typhoid, and cholera. There is swelling and thickening of the bile-ducts throughout the liver; the organ becomes enlarged; the ducts are dilated, and numerous foci of suppuration, forming smaller or larger abscesses, occur. The inflammation may extend to the pancreatic duct, and cause suppurative pancreatitis; or abscesses near the surface may lead to localised or general peritonitis. Occasionally infection extends so as to cause a general pyæmia or infective endocarditis.

The symptoms are pain and distension over the liver, loss of appetite, nausea, vomiting, pyrexia and prostration, and often jaundice. The spleen may be enlarged. The duration is from a few weeks to some months, and it is often fatal. The treatment is drainage of the ducts, where possible, by opening the gall-bladder, or any dilated ducts which may be accessible.

CHOLECYSTITIS

Inflammation of the gall-bladder itself may be catarrhal, suppurative, or gangrenous. It may co-exist with cholangitis, and is frequently the result of gall-stones (*see* p. 798). An *acute infectious cholecystitis* occurs rarely in the course of infectious diseases, especially enteric fever, but also in typhus, malaria, pneumonia, cholera, and puerperal conditions. Streptococci, staphylococci, pneumococcus, typhoid bacillus, and the colon bacillus have been found. The gall-bladder is distended, its walls thickened, and the serous coat dull, or adherent to some surrounding parts. The mucous membrane is congested or ulcerated, or presents lymph on its surface; and the contents are serum, or sero-fibrinous or purulent fluid. There is an acute onset with paroxysmal pain in the region of the gall-bladder, local tenderness, more or less resistance or even tumour, and swelling of the abdomen. The pain may extend down into the right iliac fossa, and thus lead to confusion with appendicitis. Nausea and vomiting, pyrexia generally, jaundice in about one-third of the cases, and great prostration ensue.

Treatment.—The early symptoms should be met by rest in bed, soothing local applications to the right hypochondrium, and morphia

internally. If suppuration or gangrene is suspected, or if there is good reason to believe that gall-stones are present, the gall-bladder should be opened and drained (*cholecystotomy*), or preferably in many cases entirely removed (*cholecystectomy*).

GALL-STONES

(*Cholelithiasis*)

Biliary calculi, or gall-stones, are formed from the bile in the gall-bladder, or very rarely in the bile-ducts in the liver. They vary in size from a mere sand to ovoid masses of two inches in length by an inch in breadth; more often they measure from a quarter to half an inch in diameter. They are often roughly cubical in shape, presenting facets, which indicate that several have been in contact; or they may be more rounded, or spherical. The largest have the ovoid shape, which would result from their occupying the whole cavity of the gall-bladder. They are olive-green, brown, or yellowish-brown in colour, and, on section, often show a central dark nucleus with concentric markings and radiating lines. The chief constituents of gall-stones are cholesterin, bile-pigment, and calcium salts; and the bile-pigment is mostly combined with calcium, as bilirubin-calcium. Those which consist mainly of bile-pigment are small, dark and friable. Others have a nucleus or centre of bile-pigment, and are surrounded by layers of cholesterin crystals which radiate from the nucleus; these stones are generally larger, harder, and have a paler colour. There may be only a single gall-stone of larger or medium size, or there may be several hundreds.

The prevailing view as to their formation is that they result from infective lesions of the mucous membrane of the gall-bladder, due to bacillus coli communis, or less commonly to typhoid and other bacilli. The B. coli has been found in the bile-passages in association with gall-stones, obtaining access when the flow of bile is sufficiently retarded, but not existing there normally. Typhoid fever has been an antecedent in many cases of gall-stones, and its bacillus readily invades the gall-bladder. Moreover, bacilli have been found alive in gall-stones after long periods.

An excess of cholesterin forms through the degeneration of the epithelial cells, and the desquamated cells and bacteria form nuclei upon which the crystals of cholesterin are formed.

Ætiology.—Gall-stones are commoner in advanced life, and occur in women more often than in men. Sedentary occupations and over-indulgence in food seem to have some influence; and the fatty and starchy constituents of food are thought to be more injurious. A diet likely to diminish the alkalinity of the bile has been held responsible, but the bacterial relations of gall-stone point rather to catarrhal conditions of the mucous membrane, than to chemical variations in the food.

Effects of Gall-stones.—1. They may remain in the gall-bladder for years without giving rise to marked symptoms: but they cause some dragging pain in the right hypochondrium, and according to some they are responsible for some of the symptoms classed as dyspeptic. A gall-stone or gall-stones can sometimes be felt through the abdominal wall as a hard mass, which may, in somewhat rare instances, give a sensation of crackling on being handled. In many instances, at some time or other, infection occurs, followed by catarrhal or suppurative *cholecystitis*, in which the gall-bladder may be largely distended, its wall thickened and ultimately gangrenous, with a possible opening into the peritoneum as a result. In other cases the inflamed wall of the gall-bladder contracts adhesions to the colon or the duodenum, when a fistulous communication is made between them, and the stone or stones pass directly into the intestine. Such a stone is rarely vomited: more frequently it passes *per anum*, or causes a fatal intestinal obstruction (*see* p. 742). Not infrequently, also, the gall-bladder adheres to the abdominal parietes, and an abscess is formed in them, which communicates often by a sinuous tract with the cavity of the gall-bladder.

2. In consequence of increased pressure or of some irritation in the gall-bladder, one or more stones may be forced out of the cavity and may be impacted in the cystic duct. The immediate effect is spasm of the duct and intense pain, and symptoms much like those presently described in detail, but without jaundice. The full bladder becomes distended with mucus, or muco-pus or pus: cholecystitis again ensues, with gangrene, ulceration of the gall-bladder, its adhesion to surrounding parts, and other similar changes. Sometimes a chronic impaction in the cystic ducts leads to shrinking of the gall-bladder and obliteration of its cavity.

The symptoms may be relieved by the stone falling back into the bladder, or passing on into the common bile-duct and duodenum.

3. If a gall-stone is impacted, as it may be, in the common bile-duct, the passage of bile into the duodenum is impeded, or prevented. As a consequence, bile stagnates in the bile-passages, and passes into the lymphatics and the blood, and thence the bile-pigment reaches the urine: at the same time the fæces are pale, clay-coloured or colourless. In either event an attack of *biliary colic* occurs, more certainly recognised in the latter case, because of the occurrence of jaundice from the circulation of the bile-pigment in the blood.

In an attack of biliary colic, the patient is seized, often suddenly, with agonising pain in the right hypochondrium and lower part of the chest, in the right shoulder, and back. This is often so severe that he is bent double, or writhes on the floor or bed. Shivering may occur, and even epileptiform convulsions, and the patient is pale, collapsed, with profuse sweating, and a small, feeble, generally quick pulse. After a time the pain becomes dull and aching, until a fresh attack of the acuter kind occurs; or the pain persists, and within a few hours, or a day or two of the beginning

of the pain, bile-pigment appears in the urine, and the patient becomes jaundiced. This is an indication that the gall-stone is impacted in the common bile-duct, so that an obstructive jaundice has resulted. All the signs of this condition are present—deep jaundice, bile-pigment in the urine, and pale fæces. This again may end by the passage of the stone into the duodenum, when the bile again flows freely, the pain subsides, and more gradually the jaundice clears up. When this happens, the fæces should be searched for the gall-stone, which may be found by washing them with water and passing the washings through a sieve.

But the time which elapses between the impaction and the discharge into the duodenum is very variable; it may be several weeks, during which the patient remains jaundiced, and is subject to more or less pain, the liver being also somewhat enlarged and the gall-bladder distended. Even then the stone may pass, and the patient is free from further trouble until another stone travels down the duct. In this way several attacks may occur.

On the other hand, there may be a permanent impaction of the gall-stone in the common bile-duct. This is not infrequently in the ampulla of Vater, at the termination of the bile-duct, where the very small orifice into the duodenum offers greater resistance than the calibre of the duct itself. This continued obstruction has effects on the liver, on the gall-bladder, and on the pancreas.

The liver is at first considerably enlarged from dilatation of its ducts, which are distended with bile. Sometimes the ducts are dilated uniformly, at others more irregularly into globular cysts; after a time, too, their contents become mucous in character. They exert a certain amount of pressure on the tissue of the liver, and cause it to atrophy; so that subsequently the liver becomes smaller and rather flaccid.

The effect of the impaction on the gall-bladder is to cause its distension, so that it projects below the edge of the liver, nearly in the mammary line; here it can be recognised as a large tense cyst, globular or ovoid in shape (since its fundus is the part that is felt), freely movable, unless fixed by adhesions, and descending on inspiration with the enlarged liver. The bile which it contains gradually gets mixed with mucus, secreted from its lining membrane, and ultimately mucus may be present alone. When the downward flow of the bile is impeded by an obstructing calculus, bacteria, especially the colon bacillus, usually present in the ampulla of Vater, are enabled to get up the biliary passages; and thus infective and suppurative cholangitis, and cholecystitis, with gangrene or suppuration, are enabled to occur, and may be followed by abscesses in the liver and septicæmia and pyæmia.

When a gall-stone is fixed in the ampulla of Vater, the relations of the pancreatic duct, which opens here, are of importance. The flow of bile, prevented in the direction of the duodenum, may take place into the duct of Wirsung, and this appears to be one cause of

acute hæmorrhagic pancreatitis. But bacterial organisms readily pass along the pancreatic duct in which the flow is retarded, and thus set up less acute or chronic forms of pancreatitis.

4. Another result of the presence and persistence of gall-stones in the bladder is, undoubtedly, cancer, which begins frequently in gall-bladders containing gall-stones, and ultimately spreads to the substance of the liver, the colon, or the duodenum.

Diagnosis.—Biliary colic may be confounded with other sources of pain in that region: pleurodynia, pleurisy, intercostal neuralgia, gastric pain, intestinal colic, renal colic, and appendicitis; these can generally be distinguished by the localisation of the pain or other features accompanying them. But the passage of gall-stones is not always associated with pain, and cases of impaction with jaundice may be misunderstood from the absence of this symptom. In older cases, the history of repeated attacks is of great service. The recognition of the inflammatory complications described must depend on a careful analysis of the symptoms. When the gall-bladder is distended by retention of bile, or by secretion in acute cholecystitis, it often forms a tumour of which the shape is distinctive. But when there is acute tenderness it may be difficult to localise it correctly, and its confusion with appendicitis may actually occur; in such a case one should attempt to make out the iliac origin of an appendical tumour with an upper convex border, or the hepatic origin of a gall-bladder with a lower convex border. But it may be impossible to recognise anything more definite than a rounded outline. It must be remembered that the gall-bladder is covered largely by the liver: so that a gall-bladder may be so tense as to give a sensation of stony hardness to the liver in front of it without projecting at all from its lower edge.

Prognosis.—This need not be unfavourable in a first attack of colic; many people recover even after several: but when jaundice from impaction is of long duration, the possibility of more serious consequences, such as cancer, must not be forgotten.

Treatment.—Any special treatment for the prevention of gall-stones can scarcely be undertaken before their presence is indicated by an attack of biliary colic. The diet should then be carefully regulated, moderate in quantity, starch and fat should be limited, sufficient exercise should be taken, and alkaline waters should be drunk freely. Those of Carlsbad, Vichy, Kissingen, Marienbad, and Ems are especially recommended. Supposed solvents of gall-stones (sodium sulphate, carbonate and phosphate) are of doubtful efficacy: olive oil (2 to 10 ounces daily) seems to have done good in some instances. Sodium salicylate promotes the flow of bile, and may be given in doses of 10 grains three times a day.

For an attack of biliary colic, the patient should be placed in a hot bath, or hot fomentations or poultices should be applied to the right side. Most relief will be obtained from the subcutaneous injection of a quarter to a third of a grain of morphia, repeated,

if necessary, in three or four hours; less speedy relief is given by opium, of which the dose may be 2 grains, followed by a grain every three hours till the pain is allayed. Murchison recommended half a grain of extract of belladonna every two hours; and chloroform may be inhaled with temporary relief.

Many of the results of gall-stones can be relieved surgically. The stones can be removed by opening the gall-bladder; but in many cases the wall of the gall-bladder is already much damaged, and cholecystectomy proves the best measure to adopt. Stones in the cystic duct may be removed through the incised gall-bladder. A calculus in the common duct is more difficult to remove, but it may be reached by an incision in the duct; or if soft, it may be crushed by the finger; or it may be broken up by a needle, and in either case left to be discharged into the duodenum.

PERIHEPATITIS

Pathology.—Perihepatitis, or inflammation of the capsule of the liver, may be acute or chronic, localised or more generally diffused. It is set up by several of the structural changes in and about the liver, such as cirrhosis, lardaceous deposit, syphilitic disease, gall-stones and cholecystitis, cancer, hydatid, and abscess; or by extension of inflammation from an ulcer of the stomach or duodenum. In some of the most marked, and especially the chronic forms, it is often associated with chronic peritonitis, with chronic mediastino-pericarditis, and with chronic pericarditis and pleurisy, forming, indeed, a part of the complaint described as polyorromenitis (*see* p. 818). In other cases it is accompanied by chronic interstitial nephritis. It is often difficult to find a cause in these mixed cases, but spirit-drinking is not responsible for them.

When the liver is affected with perihepatitis, the capsule is opaque and more or less thickened; often the thickening is distributed in patches irregularly over the surface; and such patches may be determined by the disease which causes the inflammation of the capsule. Sometimes the liver is completely enclosed in a membrane, or thick casing, from one or two to seven or ten millimetres in thickness (Germ. *Zuckergussleber*). In such cases the organ has a rounded anterior edge, which is due to the actual margin of the liver being bent back upon the upper surface and held there by the thick capsule. This membrane may be stripped off without destroying the tissue beneath. The liver is rarely cirrhosed, but is generally soft and often fatty. In the severer forms ascites is generally present; and this has been supposed to arise either from constriction of the portal vein in the portal fissure, or from compression of the whole organ by its thick capsule, so as to hamper the portal circulation, or lastly to be independent of the liver, and to result from the accompanying peritonitis. The spleen is often at the same time similarly affected (*perisplenitis*).

Symptoms.—The perihepatitis which arises in consequence of localised diseases of the liver, such as hydatid, abscess, cancer, or gall-stone, especially if acute in its occurrence, may give rise to severe pain, and a friction sound is heard, or the rub can be felt on laying the hand over the liver.

In the more extensive cases associated with general chronic peritonitis also, the early symptoms may be acute, with fever, pain, and malaise; but these symptoms are not always present, and the early changes may be quite insidious.

In all cases ultimately ascites occurs; the liquid is abundant and occurs again and again after paracentesis. There is sometimes a slight amount of anasarca, especially when the pericardium is involved. Jaundice is absent.

Treatment.—The pain of acute attacks may be relieved by opium and local anodynes. In the chronic cases paracentesis will require to be performed frequently.

PYLEPHLEBITIS

This occurs in two forms, adhesive and suppurative, which have been already referred to, the one as a cause of ascites (*see* p. 768), the other in connection with multiple abscesses of the liver (*see* p. 772).

ADHESIVE PYLEPHLEBITIS

This is more generally a thrombosis of the portal vein, in which the clot adheres to the wall of the vein, and becomes ultimately organised in the same way as thrombus in other situations. Its causes are those changes which bring about retardation of the blood-current in the portal vein or its distribution, such as cirrhosis, syphilitic disease, the pressure of tumours on the trunk of the vein, or its implication in perihepatitis, or chronic peritonitis near the fissure of the liver.

Symptoms.—The obstruction to the portal vein necessarily leads to symptoms closely resembling, or identical with, those of cirrhosis, viz., engorgement of the portal vein radicles, showing itself in ascites, with enlarged spleen, diarrhœa, and hæmorrhage from gastric and intestinal vessels. The collateral circulation becomes developed in the same way as in cirrhosis, and the superficial abdominal veins are commonly enlarged, relieving thus, for a time, the portal vessels. The urine is scanty. Jaundice is rare, and only occurs as a result of that which causes the pylephlebitis. The liver is generally smaller than normal.

The **Diagnosis** from cirrhosis of the liver is generally difficult. It must depend on the absence of alcoholic history, or the known presence of conditions which might cause portal thrombosis; but most of these conditions are themselves possible causes of direct portal obstruction and ascites.

Its **Treatment** is similar to that of cirrhosis.

SUPPURATIVE PYLEPHLEBITIS

This is nearly always due to infection from lesions in the abdomen—*i.e.*, the area from which the blood is supplied to the portal vein, such as ulcerative appendicitis, ulcers of the rectum, colon, or small intestines, gastric ulcer, and suppuration of the mesenteric glands, pancreas, or spleen. Nearer the liver gall-stones may cause inflammation of the portal vein branches, and the same may happen from hepatic abscess or a suppurating hydatid cyst. In the new-born the portal vein may be infected from a septic phlebitis of the umbilical vein; and rarely the lesion may be caused by direct injury.

The mischief commonly begins either in the branches or in the tributaries of the portal vein, rather than in the trunk itself, to which, however, the suppurative process may ultimately extend. The wall of the vein inflames and suppurates, a thrombus forms in the neighbourhood, breaks down into pus, and its conveyance to other parts of the vessel sets up fresh centres of thrombosis, phlebitis, and suppuration. Finally, in many cases, multiple small abscesses of the liver are formed. The liver is enlarged, soft, flaccid, and anæmic. The branches of the portal vein are filled with disintegrating thrombi, or pus, or grumous fluid; and the walls of the corresponding veins are infiltrated, or ulcerated. The spleen is enlarged, and there is occasionally peritonitis.

The **Symptoms** are nearly the same as those of multiple abscesses. There are epigastric and hypochondriac pain, fever of hectic type, rigors, sweating, vomiting, anæmia and prostration. The portal vein may be sufficiently obstructed to cause some ascites, and the spleen is enlarged, partly on this account, partly as a result of septic fever. Jaundice is often, but not always, present; and, if abscesses are numerous, there may be enlargement of the liver. The fæces are generally coloured. A typhoid condition supervenes with stupor and delirium.

Diagnosis.—The disease is easily overlooked. It may be confounded with pyæmia, septicæmia, malarial fevers, acute yellow atrophy, tropical abscess, subphrenic abscess, typhoid fever, or pneumonia. Fever with rigors, local evidences of the liver being involved, such as pain, swelling and jaundice, evidence or history of a local source of infection, and signs of portal obstruction, such as diarrhœa and enlargement of the spleen, would point to suppurative pylephlebitis. When local signs are absent, the fact of an obvious pyæmia without external wound, and without endo-carditis, might suggest some abdominal organs as the source of the sepsis.

Prognosis and Treatment.—The disease runs an acute course, is generally fatal, and can only be treated by relieving symptoms.

DISEASES OF THE PANCREAS

The pancreas is subject to pathological processes similar to those that occur in other organs. The organ itself is not easily palpable, being deeply seated, and only occasionally in thin people can it be felt lying transversely across the aorta. Even when enlarged by cancer, or chronic inflammation, it may be entirely concealed by the overlying liver. Cysts, however, of the pancreas are often large enough to form considerable tumours in the upper part of the abdomen.

GENERAL RESULTS OF DISEASE OF THE PANCREAS

The Fæces.—If the secretion of the pancreas is not poured into the abdomen, the fatty constituents of the food are imperfectly digested, and bulky, soft, pale, *fatty stools* are passed, in which an oily liquid is actually present, or lumps of white or yellow fat like tallow; and crystals of the fatty acids may be seen under the microscope (liporrhœa). This condition is not constant, as the bile assists in the solution of the fatty matters, and it has already been shown that fatty stools may occur when the biliary secretion is retained. In cases of malignant disease of the pancreas the total fat in the stools may reach 80 or 90 per cent. A clay-coloured appearance of the stools has also been attributed to absence of the pancreatic secretion, and has been explained by the view that the colouring matter of the fæces is only formed as a result of the co-operation of the pancreatic secretion with that of the liver. Stercobilin is often entirely absent in cancer of the pancreas, but present in some degree in other diseases.

In cancer of the pancreas, undigested muscular fibres are often found in the stools (*azotorrhœa*).

The Urine.—Another secretion liable to be influenced by disorders of the pancreas is the urine; and it is well known that an intimate relation exists between diabetes mellitus or glycosuria and pancreatic disease, both acute and chronic. The relation is, however, not constant, and glycosuria is not always present in cases of acute or chronic pancreatitis or cancer of the pancreas, where the symptoms of the visceral disease are prominent and call for diagnosis and relief; on the other hand, in most cases of diabetes there are no other clinical signs of local disease of the pancreas. But although sugar is not generally present, it is stated by Dr. Cammidge that in cases of pancreatitis the urine has a specific reaction. This was discovered by him in his attempts to find in that secretion, the glycerine which might be expected as a result

of fat-necrosis (*see* below); but the reacting substance is not proved to be glycerine. The test, which can only be carried out in the laboratory, is roughly as follows: The urine is examined for albumin, sugar, bile, urobilin, and indican. An estimate of the chlorides, phosphates, and urea is also made, and the deposit is examined for calcium oxalate crystals. If the urine is free from albumin and sugar, it is acidified with 5 per cent. of strong hydrochloric acid, gently boiled for ten minutes, cooled, and the excess of acid neutralised by lead carbonate. The filtrate is treated with tribasic lead acetate to remove glycuronic acid, and the excess of lead is removed by hydrogen sulphide or sodium sulphate. It is then tested with the phenylhydrazine hydrochlorate (*see* Diabetes). In well-marked cases of pancreatic inflammation a light yellow flocculent precipitate forms in a few hours consisting of long, light yellow, flexible, hair-like crystals in sheaves, which dissolve readily in 33 per cent. sulphuric acid.

Sugar, if present, must be removed by fermentation, and albumin by coagulation and filtration, before the test is applied. This reaction is regarded by many rather sceptically.

Fat-Necrosis.—A frequent result of disease of the pancreas, especially acute inflammation and hæmorrhage, is the condition known as *fat-necrosis*, which is discovered *post mortem* or during operation, and does not reveal itself as a clinical sign. In the pancreas, and in the sub-peritoneal fat adjacent, and even in the perinephric, mediastinal, and pericardial fat, are small masses of dull yellow or opaque white colour, sharply differentiated from the adjacent healthy fat. They appear to be produced by the action of the fat-splitting ferment of the escaped pancreatic secretion, of which the fatty acids combine with calcium bases.

ACUTE PANCREATITIS

Acute inflammation of the pancreas sometimes occurs in infective diseases, such as enteric fever, pyæmia, and septicæmia; and also in mumps, which is of interest from the structural resemblance of the pancreas to the salivary glands. It arises also by extension from neighbouring parts, from obstruction of the duct of Wirsung by pancreatic calculi, and by the spread of infection from biliary calculi. If a gall-stone is impacted in the ampulla of Vater, this may not only retain the pancreatic secretion in the duct, but by preventing the bile from flowing into the duodenum may force this secretion also into the pancreas.

In enteric fever pancreatitis is commonly of the parenchymatous form, the connective tissue being infiltrated with leucocytes, and the gland-cells in a state of cloudy swelling.

In *acute hæmorrhagic pancreatitis* the organ presents the usual pathological appearance of inflammation, and in addition there is hæmorrhage, often abundant, both under the capsule and into the

interstitial tissue; and the blood may extravasate into the surrounding structures.

In *suppurative* forms the organ is large, swollen, and infiltrated with pus; or it contains separate abscesses. *Gangrenous* changes may be associated with the hæmorrhagic; they often lead to peritonitis. The bacillus coli communis and pyogenic organisms are found in different cases.

Symptoms.—It is probable that the milder forms of acute pancreatitis may be the cause of some cases of catarrhal jaundice, by compression of the common bile-duct. When pancreatitis occurs in the course of mumps, there are vomiting and epigastric pain, with swelling and tenderness in the epigastric region. The more intense and hæmorrhagic forms are characterised by severe, even excruciating, pain in the upper part of the abdomen, tenderness, muscular rigidity, nausea, vomiting, and collapse. In the hæmorrhagic cases, the symptoms often come on quite suddenly in the midst of apparent health, and the symptoms have been constantly mistaken for those of intestinal obstruction, or peritonitis from perforation of a gastric ulcer, or biliary colic. Often there is nothing distinctive; sometimes, after a few hours, a circumscribed, tender swelling appears in the upper part of the abdomen, but the difficulties of diagnosis are such that the abdomen has often been opened for the relief of a supposed intestinal obstruction. The cases are generally fatal within four or five days, but some have recovered after laparotomy. The symptoms of suppurative pancreatitis are similar, but less pronounced and acute.

Treatment.—Little can be done for acute pancreatitis beyond the relief of pain and vomiting by local applications and morphia; but the diagnosis will rarely be made with certainty prior to laparotomy, when the condition that is found may suggest local measures —*e.g.*, an abscess might be opened and drained. Even with a hæmorrhagic lesion, the organ has been incised, the hæmorrhage checked by ligature, and drainage instituted with success.

CHRONIC PANCREATITIS

This affects the interstitial tissue, producing considerable fibrous growth, with consequent atrophy of the glandular structures, analogous to the changes in cirrhosis of the liver; and as in that disease, the fibrous network may enclose large groups of acini (*interlobar*), or single acini (*interacinar*). The head of the organ is usually most affected. The substance is rendered extremely dense and hard, and in the less common interacinar form considerable enlargement may take place. It commonly arises from the spread of adjacent inflammations, such as those of the peritoneum, of the bile-duct, or of a gastric ulcer. It also results from the presence of concretions in the pancreatic duct, or of retained pancreatic secretions; from the irritation of cancer; from the venous congestion of heart disease;

and possibly from syphilis, and the abuse of alcohol. Probably the most common antecedent condition is cholelithiasis, or gall-stones, especially when there is a gall-stone in the ampulla of Vater, or in the common duct, or when there is suppurative cholangitis, the result of gall-stones.

Symptoms.—These are not always prominent; and when the condition has been found it has often been at an operation undertaken to relieve some one of the various results of gall-stone, of which jaundice and epigastric pain are the most common. But apart from gall-stones and often without pain, the swollen head of the pancreas may compress the common bile-duct, which it surrounds, and thus cause jaundice and distension of the gall-bladder; and these will be associated with the characteristic stools, and possibly glycosuria or other urinary indication. Mayo Robson records cases where chronic pancreatitis, indicated by the Cammidge reaction, has resulted in glycosuria and fatal diabetes. If the body of the gland alone is involved, jaundice is absent, and the symptoms may be few or none unless the swelling is so much as to render the organ palpable.

Treatment.—As the chronic inflammation is attributable in so many cases to disordered conditions of the biliary and pancreatic ducts, the relief of these by operation is often the chief object of treatment. For this, calculi should be removed and the gall-bladder should be drained (*cholecystotomy*), or the gall-bladder may be united to the bowel (*cholecystenterostomy*).

HÆMORRHAGE INTO THE PANCREAS

This has been already mentioned as an accident of acute pancreatitis; but hæmorrhage occurs sometimes apart from inflammation, especially in conditions of the circulation, such as those accompanying heart disease and emphysema, which conduce to venous congestion of the abdominal organs. Blood-cysts may form in the substance of the gland. A few instances are on record in which hæmorrhage seemed to be the sole cause of death.

DEGENERATION

The recognised forms of degeneration are *atrophy, fatty degeneration, fatty infiltration,* and *lardaceous degeneration.* These are rarely productive of definite local symptoms; but atrophy and fatty change are not infrequently found in cases of diabetes. The destruction of the cells of Langenhans by the degenerative process has been thought to be essential for the occurrence of diabetes, but this is open to doubt.

PANCREATIC CONCRETIONS

These may occur in middle-aged men; they are by no means common. They are attributable to catarrh of the ducts with delayed secretion, and consist of calcium carbonate and calcium phosphate, and sometimes calcium oxalate. They may be like grains of sand, or as large as hazel-nuts, and are usually round or oval, occasionally irregular or branched. In colour they are white, or grayish white; sometimes brown or nearly black. They sometimes block the duct or its branches, and lead to dilatation of the ducts, retention-cysts, acute inflammation with suppuration or chronic induration, and even to inflammation in the parts around. They rarely produce symptoms, except through their secondary effects—for instance, by the inflammation which they excite, or by the formation of cysts, or by the production of atrophy and cirrhosis of the gland.

TUMOURS OF THE PANCREAS

Of these, *carcinoma* is the most important, whether primary or secondary. Sarcoma, tubercle, gumma, and lymphoma only occasionally occur. Primary cancer is generally of the scirrhus variety, forming hard nodules in the head of the organ, to which part it is often confined. An irregular nodular hard tumour is thus formed, which may be of sufficient size to be felt under favourable circumstances through the abdominal parietes. As the cancer nodules increase in size the pancreatic duct is liable to be obstructed, with the formation of a cyst as a result; and the common bile-duct is not infrequently blocked either by pressure or by the spread of a chronic inflammation, so that jaundice is produced. This is, indeed, a common cause of jaundice in persons of middle and advanced age. In other instances the cancer may involve the stomach, duodenum, peritoneum, vertebræ, or other structures. The symptoms are variable; pain may be absent, but it is sometimes deep-seated, of aching, gnawing, lancinating, or burning character, often distinctly paroxysmal in its occurrence, and affected by food, coughing, deep breathing, movement, or posture. Nausea and vomiting may be present; and the stools are often fatty or contain undigested muscular fibres. Examination may reveal a tumour, of the characters described, in the situation of the head of the pancreas. In the later stages emaciation, anæmia, and prostration become prominent features of the case.

Pancreatic Cysts.—These are usually the result of obstruction of the duct of Wirsung by calculus, or by pressure from without: they may reach a considerable size and hold many pints of fluid. Such a cyst forms a globular tumour in the upper part of the abdomen, either in the median line, or in the left hypochondrium.

At first behind the stomach and colon, it may, if it is large, press the hollow viscera aside. A pancreatic tumour is often stationary during deep inspiration, but it may move downwards half or three-quarters of an inch. The surface is dull or resonant, according to the extent to which it is covered by either of these hollow viscera. The fluid within it is turbid, brown or greenish in colour, alkaline, of sp. gr. 1010 to 1020; it contains albumin, sugar, mucin, and a trace of urea; and it has the property of digesting starch and emulsifying fat. Hæmorrhage may take place into it. There is emaciation, and sometimes pain or jaundice. The swellings most likely to be confounded with it are a hydatid cyst of some other organ, hydronephrosis, circumscribed peritonitis, and ovarian disease: but if much to the left and moving on inspiration it may resemble a splenic or renal tumour. The nature of the aspirated fluid should help. Congenital cysts and hydatid cysts of the pancreas occur rarely.

Treatment.—Pancreatic cysts have often been successfully treated by incision and drainage. Other tumours are less easily dealt with; and treatment must be directed to the relief of symptoms. If the tumour cannot be removed, some relief may be obtained by cholecystotomy or cholecystenterostomy.

DISEASES OF THE PERITONEUM

PERITONITIS

The peritoneum lining the surface of the abdomen, and covering nearly all the viscera contained within it, is liable to inflammation from a number of causes originating in these organs as well as from more widespread infection. This inflammation may be acute or chronic, and general or circumscribed.

ACUTE PERITONITIS

Ætiology.—The most frequent cause is some lesion of the abdominal viscera or adjacent parts, such as ulceration of the stomach, typhoid and tubercular ulcers of the ileum, dysenteric ulcers of the colon, inflammation and sloughing of the appendix cæci, abscess of the liver, suppuration of the gall-bladder, infarction and abscess of the spleen, the numerous inflammatory lesions which are apt to involve the female pelvic organs—metritis, parametritis, ovaritis, salpingitis, and pelvic hæmatocele.

In many of these cases the peritonitis is set up, not so much by extension of inflammation, as by the discharge into the abdominal cavity of liquids, such as food, fæces, or pus, which owe their irritating properties in large part to septic organisms. This happens

in the case of the perforation of gastric and intestinal ulcers, in appendicitis, and in rupture of abscesses. Peritonitis is the natural termination of most cases of intestinal obstruction, either from local inflammation, as in acute strangulation and hernia, or from rupture of an over-distended gut, as in the more chronic strictures. Perinephric and psoas abscesses may rupture into the peritoneum, and empyema occasionally sets up inflammation below the diaphragm, though it is much less common than pleurisy and empyema as a result of a peritoneal abscess. Wounds of the peritoneum, whether from injury or surgical procedure, are liable to be followed by peritonitis.

Infection of the peritoneum from the blood apart from local lesions is less common. A pneumococcal infection sometimes occurs first in the peritoneum, but even then perhaps it may be transmitted by the pelvic passages; and much more often a pneumococcal peritonitis is secondary to pneumonia or empyema. Gonococcal peritonitis is generally associated with vaginitis. And if peritonitis appears to form part of a general septicæmia or pyæmia, puerperal or otherwise, it may nevertheless arise directly from a local lesion. Bright's disease, whether acute or chronic, is an occasional cause of peritonitis, which is then generally fatal.

Morbid Anatomy.—The changes which take place in the peritoneum are not unlike those which occur in the pleura when it is inflamed. There is at first redness from increased vascularity, and if the cavity of the abdomen is examined in this early stage, the redness of the intestine is commonly seen to be most marked along two parallel lines, which are determined by the diminished atmospheric pressure in the space between any two coils and the abdominal wall (hence called *suction-lines* by Moxon). This injection is soon followed by the effusion of lymph or pus. There is at first a mere stickiness of the peritoneal surface, but the lymph soon becomes more abundant, forming yellow flakes, coating the surface of the bowel, or collecting in larger masses in the angles and sulci between the coils. With this lymph, which consists of fibrin and leucocytes, there may be a varying amount of turbid serum. This forms with great rapidity, as may be seen in some traumatic cases, where a quantity of yellow lymph may be formed in less than eighteen hours. In some less severe or less extensive cases, the lymph may develop into fibrous tissue, by the growth of some leucocytes into fibres, and of others into blood-vessels, and the different viscera are united together, or the peritoneal cavity is obliterated, by the adhesions which are thus formed.

In other cases, the quantity of leucocytes increases, or is more numerous, from the first, and the inflammatory products are entirely purulent: this is often quickly fatal, but occasionally a large peritoneal abscess may slowly form and offer chances of recovery.

Acute peritonitis is sometimes, from the first, circumscribed, and results in a localised abscess, which may point externally or open into one of the hollow viscera. Such abscesses occur in the pelvis, or between the diaphragm and the liver, or between the diaphragm

and the spleen. In these last two situations they may rupture into the chest, and set up pleurisy or pneumonia. A peritoneal abscess not infrequently contains air, either from direct communication with the stomach or intestine by perforation of an ulcer, or from decomposition induced by contact with the coats of the bowel and transference of micro-organisms.

Bacteriology.—The micro-organisms causing peritonitis are usually the bacillus coli communis when the peritoneum is infected from the intestine, as in appendicitis or perforation of the bowels, or from the biliary passages; streptococci and staphylococci are found in peritonitis derived from lesions of the pelvic organs, or the abdominal walls. But the amœba coli has been found in amœbic dysentery; and the pneumococcus and gonococcus are also causes of an acute peritonitis. The tubercle-bacillus occasionally causes acute inflammation, but much more commonly a chronic form (*see* p. 815). The infections are often mixed.

Symptoms.—*Acute general peritonitis* begins with pain, which is mostly very severe, and, if at first localised to one spot, soon becomes diffused over the whole abdomen. The pain is constant, but aggravated by every kind of movement, by coughing, straining, or vomiting. It is not relieved by pressure; on the contrary, there is marked tenderness over the whole of the abdomen. Vomiting, as a rule, soon sets in, and occurs repeatedly, either spontaneously or after attempts to take food. At first the gastric contents are brought up, subsequently bile, and later still, in some prolonged cases, the vomited matters may have an almost feculent character. The temperature commonly rises, reaching 102° or 103°, more rarely 104° or 105°; and the pulse is quick, 100 to 120. Sometimes, also, rigors occur at the commencement; but there is always a considerable degree of collapse.

In some cases of perforation of gastric or intestinal ulcer, death occurs from collapse in twenty-four hours. In others, the patient is soon obliged to take to his bed, and within a short time lies on his back, with shrunken face, dark sunken eyes, anxious expression, dry furred tongue, and quick small pulse. The legs are frequently drawn up to prevent stretching of the abdominal parietes, and every movement is avoided by the patient. The abdomen is at first tense, with rigid muscles, and immobility during respiration. Later it becomes swollen from paralysis of the muscular coat of the intestines, and the accumulation of gas within them. The surface is resonant, but if much fluid is poured out, it may cause dulness at the flanks, or occasionally all over. Gas may escape into the peritoneal cavity, and cause extensive resonance, or even splashing from mixture with the liquid. The extravasation of gas into the peritoneum is sometimes recognised by its lying in front of the liver, and replacing the natural hepatic dulness by resonance. But it must be remembered that the liver may be displaced from its contact with the anterior thoracic wall by much gaseous distension of the viscera, without any escape of gas from their interior. The

tension of the abdomen causes pressure on the under surface of the diaphragm, with consequent dyspnœa, and generally the respiratory movements are entirely thoracic. Hiccough is also a frequent symptom. The bowels are, as a rule, confined; sometimes, after two or three days, one or more motions may be passed, or even diarrhœa may set in; and occasionally there is diarrhœa from the first. The urine is scanty; it may be passed with pain, or be retained.

The patient gradually gets exhausted by vomiting and pain; the tongue becomes drier and brown; sordes form on the lips and teeth; the pulse is smaller and quicker; the bases of the lungs are compressed; and after an illness of from two to six days, death takes place. It is not, however, every case that presents all the characteristic signs. Fever is absent in some cases; there is but little distension in others; occasionally, a patient, instead of lying prostrate on his back, will throw himself about in the agony of pain.

In *acute circumscribed peritonitis* (of which many cases of appendicitis are examples), the symptoms are much the same, but the local conditions are more or less limited to the region affected. If pus forms, a more or less defined tumour may be recognised, and fluctuation may be detected. The constitutional symptoms are often less severe than in general peritonitis; and the case may possibly run on into a chronic stage. The fever is variable. The latter course of the illness is determined by the behaviour of the inflammatory products; a sero-fibrinous peritonitis may subside; a peritoneal abscess may burst into different cavities, or set up inflammation in the chest, in which case the symptoms of pneumonia, pleurisy, empyema, or pneumothorax may complicate the abdominal lesion, and a fatal result is rendered highly probable, but not absolutely certain. Disease of the appendix cæci, and lesions of the uterus and its appendages, are the more common causes of acute circumscribed peritonitis.

Subphrenic pyo-pneumothorax is the condition which results from a local peritonitis in the upper part of the abdomen, accompanied by the escape of air into the peritoneal cavity. The most common cause is the perforation of an ulcer of the stomach; hence the abscess is mostly on the left side. The air-containing cavity then lies between the left lobe of the liver and the left half of the diaphragm, and is bounded on the right by the falciform ligament, and in other directions by the anterior abdominal wall, the stomach, and spleen. On the right it is between the upper surface of the liver and the diaphragm, and is limited to the left by the falciform ligament. The cavity contains pus and air; the liver and spleen are depressed, and the diaphragm is pushed upwards so that a pleural pneumothorax is simulated by the occurrence of tympanitic note, amphoric breathing, metallic tinkling, and bell-sound.

Diagnosis.—As a rule, this is not difficult: the severe pain, tenderness, vomiting, rigidity, and immobility of the abdomen

during respiration, followed by distension, constipation, small quick pulse, and collapse, form the important features. But peritonitis may be simulated by the severe pain of colic, by hysteria, by ruptured aneurysm, and by acute hæmorrhagic pancreatitis; it may itself be mistaken for intestinal obstruction; and it may be set up and cause death without its presence being suspected, in enteric fever, and after operations on the abdominal walls, such as herniotomy. *Colic* and *hysterical pain* are mostly to be distinguished from peritonitis by the contracted abdomen and the absence of tenderness—indeed the relief on pressure, in the former case; and the extreme sensitiveness to the merest touch, without pressure, in the latter. Indications of lead-poisoning in the former, or a history of hysterical attacks in the latter, would assist the diagnosis. A high temperature or extreme collapse is in favour of peritonitis. A *ruptured aneurysm* causes pain and collapse, and may be mistaken for the perforation of a gastric ulcer; and the antecedents in the two cases may be difficult to discriminate. Perforative peritonitis is sometimes simulated by the fatal coma or collapse of *diabetes*. This often begins suddenly, with severe abdominal pain accompanied by collapse, and small, thready pulse. Most of these cases are known to have diabetes, or a history of its symptoms can be elicited. Peritonitis resembles *intestinal obstruction* in its pain, distension, vomiting, and constipation, which may hamper the diagnosis for two or more days; the generally diffused tenderness, early distension, and simple gastric or bilious vomiting, are in favour of peritonitis; and this is often at length confirmed by the passage of fæces. The onset of peritonitis in typhoid fever may be very insidious; the patient is perhaps semi-comatose or delirious, and his senses are dulled; the abdomen is already much distended and tense. On the other hand, the tympanites and pain of this fever may be thought to be due to peritonitis when none exists.

As to the differential diagnosis of peritonitis, its cause must be looked for in the preceding history. Where severe acute peritonitis ensues in a person previously considered well, ulceration of the appendix cæci, perforating gastric ulcer, and lesions of the pelvic organs are the most likely causes. The first is more probable in both sexes before and about the age of puberty; the last occurs almost exclusively in females, and in girls the possibility of a neglected vulvo-vaginitis causing gonococcal peritonitis should be thought of. Pneumococcal peritonitis may also be apparently spontaneous and primary. Perforations of the intestines, whether typhoid, tubercular, or dysenteric, are generally preceded by recognisable illnesses; but, as is well known, typhoid fever may be of such a mild nature as not to attract sufficient attention on the part of either the friends or the medical man.

Subphrenic pneumothorax has to be distinguished from *pleural pneumothorax*. In the former there is often the history pointing to gastric disease, there is no cough or expectoration, the heart is only slightly displaced and that upwards, there is no intercostal

bulging, the liver and spleen are less depressed, and the diaphragm in some cases moves freely. The diagnosis from *gaseous distension of the stomach* by physical signs is difficult, but the associated conditions are different.

Prognosis.—General peritonitis is a very fatal disease. The probable result must be estimated by the character of the pulse, the persistence of vomiting, the amount of collapse, and the probable extent of the inflammation. Severer cases can only be judged of from day to day. There is more hope when some days have elapsed, but in cases that are apparently improving, accumulations of pus may reveal themselves, and become dangerous in the way indicated. Pneumococcal and gonococcal forms are relatively favourable.

Treatment.—The majority of cases of peritonitis, and especially those due to perforation of a gastric, duodenal, or typhoid ulcer, or sloughing of the appendix cæci, or other similar accident, are only likely to recover if promptly treated by surgical methods—the abdomen must be opened, the cavity washed out, and the causative lesion dealt with. The same may be said of general suppurative peritonitis, or localised peritoneal abscesses. One of the first considerations then is, whether the case is one of this kind.

Moreover, if the origin of the peritonitis is doubtful and the patient is acutely ill, it is safer to do an exploratory laparotomy, and deal with whatever lesion is found, than to delay until a fatal septic condition is established. Exceptionally a peritonitis of slight extent and less acuteness may be justifiably treated by other means. The first principle of treatment is to keep the intestines completely at rest. For this purpose the patient must, of course, be in bed; food should be given by the rectum; and purgatives should be strictly avoided. The patient's thirst may be quenched by small pieces of ice sucked from time to time, but no food should be allowed by the mouth; the enemata may be peptonised beef-tea, milk and egg to the extent of four ounces given every four hours, or nutrient suppositories may be employed. Opium may be given in doses of one grain every four hours; or morphia may be used instead of opium, one-third or half a grain being injected subcutaneously at first, and doses of a quarter of a grain from time to time so as to control the pain.

Relief is afforded locally by the application of hot linseed-meal poultices, or flannels wrung out of hot water and sprinkled with turpentine or liniment of belladonna. Ice compresses or pieces of ice between layers of flannel are sometimes used, but they do not generally give so much relief as the hot applications. If there is much distension with gas it may be possible to draw some off by passing a long tube into the rectum. Stimulants are, as a rule, required, and are best given in the form of brandy, in small quantities frequently.

CHRONIC PERITONITIS

This may arise as a sequel of acute peritonitis, especially in its local forms; it is often the result of local irritation about particular organs—for instance, the liver or the spleen may be surrounded with a thickened capsule (perihepatitis, perisplenitis); or it may occur after repeated tappings for ascites. The growth of tubercle and cancer in the abdominal cavity gives rise to forms of chronic peritonitis, which will be spoken of shortly. Often it is impossible to explain its occurrence, but it is assumed that it is due to some form of infection (*see* Perihepatitis, Polyorromenitis). Bright's disease appears sometimes to supply the toxic agent.

Morbid Anatomy.—Chronic peritonitis, in its lesser degrees, causes no more than thickening, with opacity of the peritoneum; in more advanced cases, it results in the formation of false membranes, or layers of fibrous tissue, which cover the different viscera, and cause them to become adherent to one another, and to the abdominal wall (*chronic proliferative peritonitis*). The intestines may thus be matted firmly together, and bound down towards the spine; the omentum is shortened and contracted into a transverse band at the upper part of the abdomen; and the liver and spleen may be covered with a thick adventitious membrane. (*See* Perihepatitis.) This membrane can sometimes be peeled off, leaving the natural serous layer beneath. Serous fluid may be present at the same time in sufficient quantity to cause enlargement of the abdomen; or there may be chronic effusion, with but little false membrane, only some opacity or pigmentation of the peritoneum. As already mentioned, chronic peritonitis may result in intestinal obstruction.

Symptoms.—These vary with the extent of the abdominal lesion. There is usually some pain, or a sensation of tightness and oppression. The appetite is likely to be indifferent, and occasionally vomiting may occur. The bowels are generally constipated. Constitutional disturbance may be but slight, but fever, if present, is variable, and often the patient is about, though unequal to much exertion. The condition of the abdomen depends on the nature of the effused products; if serum exists in any quantity, the abdomen is enlarged, and gives the sense of fluctuation on percussion. Sometimes there is dulness in the flanks, and resonance in front, with change of relative position of these signs when the patient lies on one side; in other cases the abdomen is completely dull from the intestines being bound down to the spine. When there is little or no fluid the abdomen may be flat, and it presents irregular resistance where the intestines are matted together.

Diagnosis.—Cases with much effusion resemble ascites, such as that produced by hepatic cirrhosis; in women, if the surface is completely dull from the intestines being bound down, the peritoneal fluid may be mistaken for that of an ovarian cyst; in other cases,

the resistance felt in the abdomen may be confounded with different forms of tumour.

Prognosis.—This is, on the whole, unfavourable in cases with pronounced symptoms.

Treatment.—There is not the same necessity for absolute quiet on the part of the intestines as in the case of acute peritonitis; food should be light and nutritious; pain may be relieved by poultices and hot fomentations, and by opium in small doses. The absorption of the effused products may be attempted by the application to the abdomen of mercury in the form of compound ointment, liniment, or oleate; while iodide of potassium and iodide of iron may be given internally, with general tonics, such as quinine, cod-liver oil, or arsenic. Constipation may be relieved by enema, or by the mildest laxatives. Paracentesis is often necessary and may have to be repeated.

TUBERCULAR PERITONITIS

The actual inflammation in this case is preceded by the formation of tubercle as a result of the invasion of the bacillus. The surface of the peritoneum is covered with small, flat, whitish grains, from 2 to 5 mm. in diameter, slightly elevated above the surface, and closely aggregated together. These tubercles are most abundant on the under surface of the diaphragm and in the flanks. The inflammation is shown by the effusion of lymph, or of serous or serofibrinous liquid. In very *acute* cases lymph alone may be present. In less acute cases there is liquid (*ascetic* form), which may amount to several pints, and the abdomen may be as much enlarged as it is in ascites from cirrhosis or heart disease. More rarely, the liquid is sero-purulent or purulent. In other more chronic cases the results are varying degrees of caseation, fibrosis (*adhesive* or *fibrous* form), and ulcerative destruction. Thus, the omentum tends to become contracted into a thick mass, consisting of tubercular and caseous infiltration. The intestines are matted together and adherent to the omentum or the abdominal wall, and the adhesions may be much infiltrated, so as to present thick masses of almost tumour-like new tissue. When the bowel also is ulcerated, the adherent intestines may open into one another through tbe bases of the ulcers, and render it impossible to trace the natural course of the alimentary canal. More or less turbid serum or pus may be present in these cases; and the mesenteric glands are often caseous (*see* p. 867).

Ætiology.—Tubercular peritonitis occurs at all ages, but appears to be more frequent in males than in females. It is very commonly associated with tubercle in other parts of the body. It is hence often secondary to pulmonary phthisis, to tubercular ulceration of the intestine, to caseous mesenteric glands, and to diseases of the pelvic organs—*e.g.*, the Fallopian tubes, or the testes and vesiculæ seminales. It may form part of an acute general tuberculosis.

Sometimes, perhaps, the peritoneum is infected directly from the bowel. Not infrequently the pleura and the pericardium are simultaneously affected (*see* Polyorromenitis).

Symptoms.—The symptoms are sometimes *acute* and the case is similar in every respect to acute peritonitis from other infections. More often they resemble those described under *chronic* peritonitis. They have come on slowly and insidiously, consisting of pain and discomfort, and frequently distension of the abdomen. The patient loses strength and flesh; there is irregular pyrexia; the appetite is diminished, and the bowels are irregular, but often loose. The motions are sometimes yellow, and in acuter cases may suggest enteric fever. In the ascitic form with much fluid the abdomen will be enlarged, tense, resonant in its upper part, or dull all over, according as the intestines are free to float or bound down to the spine. In the adhesive form the abdomen is tender, slightly swollen, very tense, presenting to palpation increased resistance in certain parts, or a doughy sensation; or there are firm rounded masses with a more or less definite outline. Such tumour-like masses often occupy the lower half of the abdomen, reaching perhaps higher on one side than on the other: they are irregular or nodular on the surface. Sometimes the indurated masses of tubercular infiltration are felt as bands running across the abdomen; thus, the thickened omentum often forms a transverse band at its upper part, and the tissue about the obliterated urachus forms a vertical band below the umbilicus. In many cases the skin around the umbilicus, for a distance of from one to three or four inches, is reddened and infiltrated, pitting slightly on pressure: this is probably due to obstruction of the veins by the tubercular growth. The percussion resonance is variable, and often muffled. Occasionally the abdomen is retracted from the excess of fibrous tissue and consequent contraction.

Diagnosis.—Besides the general features of pain, distension, and emaciation, the recognition of the tuberculous masses and the inflammatory redness about the umbilicus are the most important. The infiltrated omentum may be mistaken for the lower part of an enlarged liver, but the resonance of the stomach above it should prevent this error. Sometimes, but not always, the diagnosis may be confirmed by the presence of tubercle in other parts of the body. In children or young people, a simple ascites, otherwise unexplained, is likely to be tubercular, but it is often difficult to distinguish from the ascites of hepatic cirrhosis, which indeed sometimes co-exists; and it has often been mistaken for an ovarian cyst until operation has proved the contrary. The liquid withdrawn by paracentesis may be tested by inoculation into animals; or tuberculin and opsonic methods may be used (*see* p. 535).

Prognosis.—This is more hopeful than the prognosis of many other tubercular lesions, and many patients treated early have apparently recovered completely. Not only does liquid become absorbed, but large masses of induration, infiltration, or matting

have disappeared entirely in some cases. Death results from exhaustion, or from acute tubercular inflammation in the pleura, pericardium, or meninges.

Treatment.—This may be conducted on the same principles as that of chronic peritonitis. Mercurial applications should be constantly applied for weeks or months. Cod-liver oil and general tonics should be used internally. The diet should be easily digestible, and stimulants in moderate amount may be given. Many cases have been treated by opening the abdomen, washing out or sponging the surface of the peritoneum, and inserting a drainage-tube. Success has attended this method, but in view of the frequent recovery without operation, the value of surgical methods is difficult to estimate.

POLYORROMENITIS

The names *polyorromenitis* (ὀρρός, serum) and *polyserositis* are given to the condition of simultaneous inflammations of two or more of the four great serous membranes, pericardium, pleuræ, and peritoneum: an association to which attention has been especially called by Italian writers. It is the result of a common infection, whether it commences in the membranes simultaneously or affects them successively, and this last is the more usual event.

It may be acute, subacute, or chronic. Acute polyorromenitis is seen in acute rheumatism, in pyæmia, and septicæmia, and as a result of pneumococcal invasion. Subacute and chronic polyorromenitis are often the result of tubercle, and the recognition of this fact is an important element in the diagnosis of some tubercular lesions. The successive invasion of the membranes may take place in almost any order, but the most frequent event is that the peritoneum is first attacked, and then the pleuræ, beginning generally with the right pleura. Some cases of indurative mediastino-pericarditis (*see* p. 664), though certainly coming under the definition Polyorromenitis, appear to be possible exceptions to the tubercular origin of chronic cases. The prognosis of the combined lesions is obviously worse than when one membrane is alone involved. (*See also* Perihepatitis *and* Chronic Peritonitis.)

PERITONEAL ADHESIONS

Some importance attaches at different times to the adhesions which form between adjacent viscera or parts of viscera covered with peritoneum, as a result of acute or chronic inflammation.

Perigastric adhesions have already been mentioned as the result of gastric and duodenal ulcers. They occur also in connection with cancer of the stomach, with operations on this organ, and with gall-stones or inflammation of the gall-bladder. They are no doubt in most cases latent, but they give rise sometimes to troublesome pains, or interfere with free movement and function of the parts

concerned. The pains are situated in the epigastric or hypochondriac region; they are variable, in some cases determined by distension of the stomach, in others occurring when it is empty; if on the posterior surface they may be relieved by the recumbent position, if in front by the converse. In other cases lateral positions may determine or relieve them; or stretching the arm above the head may drag upon them and so cause pain. It may be difficult to discriminate between the pain of adhesions and the pain of the associated lesions. In some cases relief may be obtained by operation, either freeing the organs or dealing with the original disease.

Adhesions in connection with the bowel are found in the great majority of all persons of all but the youngest ages (Mollison and Cameron), and are most frequent about the colon, and especially the ascending colon and hepatic flexure. They have been regarded both as the result and cause of constipation, but from the above observations, taken with the fact that overloading in constipation does not affect the cæcum and ascending colon, Hertz regards such adhesions as having but little importance in relation to constipation. Adhesions resulting from pelvic peritonitis, and fixing the pelvic colon, more often contribute to produce this condition. Division of the adhesions, with fixation of the pelvic colon to the abdominal wall, if necessary, has been successful in some of these cases.

NEW GROWTHS IN THE PERITONEUM

One of the most common growths in the peritoneum is *carcinoma*, secondary to disease in the viscera, especially the stomach and the ovary. It is more common in females than males, and occurs mostly at an advanced age. It occurs in the form of flat circular deposits, covering the abdominal surface, and, like tubercle, it is most abundant on the diaphragm and in the flanks; similarly the omentum may be thickened and infiltrated, and eventually large cancerous nodules may occur all over the abdomen. In its structure it is generally a true carcinoma, very fibrous, and yielding but little juice. *Colloid cancer* is present in a certain number of cases. Considerable liquid effusion is commonly present (*cancerous peritonitis*), and blood is not infrequently mixed with it, so that it acquires a brown, brownish-red, or even red colour. Occasionally nodules of cancer are felt in the skin around the umbilicus, and the glands in the groin may be infiltrated with the same growth.

Sarcoma is another common form of malignant disease: it occurs in the retro-peritoneal tissues, in the omentum, mesentery, or broad ligament.

Fibroma, lipoma, hydatid, dermoid, and other *cysts* of less certain nature also occur.

Symptoms.—The more rapid and malignant forms of growth are characterised by pain, emaciation, cachexia, and the presence of the tumour; if inflammatory conditions predominate there will

be a resemblance to other forms of chronic peritonitis. In other cases the intestine may be involved (*see* Intestinal Obstruction).

Prognosis is absolutely unfavourable, and **Treatment** must be directed to the relief of symptoms, the temporary removal of fluid when it is considerable, or an operation for obstructed bowel.

ABDOMINAL TUMOURS

It may be well to allude in this place to the great number and variety of tumours which may be found in the abdomen, and the diagnosis of which often presents considerable difficulties. Every one of the contained viscera may give rise to tumour, and to describe all these would be only to repeat what has been already said—or will be said—in the accounts of diseases of special organs; and their differential diagnosis must be gathered from the same source. It will be sufficient here to enumerate the more important tumours, and subsequently to describe one which has not been dealt with elsewhere—namely, aneurysm of the abdominal aorta.

The tumours are as follows: *Liver:* general enlargements, abscess, cancer, hydatid, distended gall-bladder; *spleen:* enlargements, abscess, cancer, hydatids, infarcts; *stomach:* cancer, dilatation; *intestines:* cancer, lymphoma, strangulation, intussusception, appendicitis, matting by tubercular disease, actinomycosis, fæcal accumulation; *pancreas:* chronic inflammation, cyst, cancer; *kidneys:* enlargements, movable kidney, abscess, cancer, hypernephroma, hydatid, hydro- and pyo-nephrosis, tubercular pyelitis; *suprarenal capsule:* cancer, tubercle; *peritoneum:* chronic peritonitis and matting, tubercular peritonitis, localised suppurative peritonitis, cancer, hydatid, and other cysts; *lymph-glands:* tubercle, lymphadenoma, cancer; *uterus and appendages:* pregnancy, fibro-myoma, cancer, cysts of the broad ligament, hæmatocele, pelvic cellulitis, ovarian and parovarian cysts and tumours; *bladder:* retention of urine.

ABDOMINAL ANEURYSM

Ætiology.—This has much the same causation as aneurysm in other situations. It is common in middle age, and is more frequent in males than in females.

Pathology.—The usual seat is between the diaphragm and the origin of the superior mesenteric artery, and it often involves the origin of the cœliac axis. In its growth it may interfere with adjacent organs, press upon the vena cava, or erode the vertebræ. Aneurysms of the superior mesenteric, or of the iliac arteries, are less common, and will not be specially considered here.

Symptoms.—These are pain, the presence of a pulsating tumour, with murmur, and sometimes evidences of pressure. The *pain* is situate in the abdomen, is often severe, paroxysmal or neuralgic in character, and may radiate to either side, into the

groin or the back. The *tumour* varies, of course, with the seat of the lesion; it is more common in the epigastric region, in the middle line or slightly to the left; it is globular or ovoid, pulsatile, and expansile; it is scarcely, if at all, affected by the movements of the diaphragm. A systolic murmur can generally be heard over it. The pressure signs other than pain are not common, since the several organs readily yield to its progress. But it may exceptionally cause jaundice by obstructing the bile-duct, or dropsy by pressure on the vena cava. It may press on the colon, or on the stomach, and vomiting is sometimes present. The duration may be two or three years, and death results, as a rule, from rupture of the sac into the retro-peritoneal tissue, into the peritoneum, or into one of the hollow viscera; or from the exhaustion of pain, sleeplessness, sickness, and malnutrition.

Diagnosis.—Abdominal aneurysm has to be diagnosed from the *excessive pulsation* of the aorta previously described (*see* p. 659), and from tumours lying in front of the aorta, especially *cancer of the stomach*, or less commonly, cancer about the *gall-bladder*, to which pulsation is communicated from the healthy aorta. The former is distinguished by the absence of murmur, the normal size and shape of the vessel, and the neurotic or hysterical character of the patient. Tumours over the aorta do not expand laterally, and are often irregular or nodulated in shape; the pulsation in some cases ceases when the patient is placed prone, or on his hands and knees, so that the tumour may fall away from the aorta. Cancer of the stomach is displaced more than an aneurysm by a deep inspiration.

Treatment.—This must follow the lines indicated under the head of Thoracic Aneurysm (p. 651). But an abdominal aneurysm is sometimes open to treatment by proximal or distal compression by the tourniquet, or other surgical means.

GLÉNARD'S DISEASE

Presenting some resemblance to abdominal tumours are the various conditions of proptosis, or downward displacement of the abdominal viscera, which go by the name of *Glénard's disease*, or *Enteroptosis*, or *Splanchnoptosis*.

This occurs especially in women, and is shown best in the upright position, when the part of the abdomen below the umbilicus is relatively prominent and the part above is flat; a condition which would be explained by a relaxation of the abdominal muscles, as well as of the internal visceral ligaments and connections, allowing the viscera to fall by their own weight into the lower part of the abdomen. When the patient lies on her back a partial restoration to the normal takes place. In extreme cases the proptosis may affect the liver, spleen, kidneys, stomach, and intestines. The movable or prolapsed kidney (*nephroptosis*) has been long recognised, and will be described later. According to Glénard, it is only a part

of a general enteroptosis or splanchnoptosis. *Gastroptosis* depends for its recognition on the determination of the position of the lesser curvature of the stomach; if this is visible below the ensiform cartilage, gastroptosis is present. Assuming that the stomach is considerably prolapsed, the colon and small intestines must be pushed down before it. Low positions of the liver (*hepatoptosis*) and of the spleen (*splenoptosis*) are also sometimes recognised.

In slighter degrees this condition may have little importance. Many women have fallen kidneys without knowing anything about it; and the same with moderate degree of proptosis of the stomach and bowels. But in others there are disturbances of digestion, which can be readily understood to result from the altered mechanical relations of the digestive tube—*e.g.*, nausea, pain after food, vomiting, and constipation; and with these are associated various nervous symptoms, partly the sensations of depression and languor, which are the intelligible result of the above digestive disturbances, partly the sensation of the dragging of the viscera upon the internal structures. Treves calls attention to the frequency with which pain and tenderness are observed at a spot a little to the left of the median line, and just above the umbilicus. The symptoms are often relieved by the recumbent posture, or by supplying a mechanical support, such as a belt round the lower part of the abdomen.

Treatment.—Little may be necessary in milder cases, though even there some strengthening of the abdominal muscles, by gymnastic exercises, may be valuable. In more pronounced cases, a belt should be worn continuously during the daytime; and, in extreme instances, the liver or the kidneys may be fixed by operation in a more natural position.

DISEASES OF THE BLOOD, DUCTLESS GLANDS, AND LYMPHATIC SYSTEM

DISEASES OF THE BLOOD

EXAMINATION OF THE BLOOD

In relation to disease, we have to study especially the total numbers of the corpuscles, red and white, details as to the kind of corpuscles, and their relative proportions, and the amount of hæmoglobin. The absolute quantity of blood in the body is also of interest.

Quantity.—The average amount of blood in the body is estimated at one twenty-second part, or 4·6 per cent. of the body-weight (Haldane and L. Smith). After any loss of blood the vessels absorb liquid rapidly from the tissues, and thus are soon filled again with a blood the same, or nearly the same, in quantity as before, if deficient in corpuscles and chemical constituents. In pernicious anæmia the quantity is often, but not always, below the normal; but in dropsy and chlorosis the quantity is increased.

Enumeration of Corpuscles, or Blood-Count. — This is effected by the *hæmocytometer* of Thoma-Zeiss or that of Gowers, which differ only in detail.

The former consists of a glass slide on which a "cell" is constructed one-tenth of a millimetre in depth and ruled at the bottom into squares, measuring $\frac{1}{20}$ mm. in the side, which are again ruled into groups of 16. In a specially constructed pipette the blood is diluted to the extent of 1 to 100 by a saline solution (sodium phosphate or chloride) which does not injure the corpuscles; and a drop of the dilution is placed in the cell and covered with thin glass. The corpuscles settle down upon the squares, each of which corresponds to $\frac{1}{4000}$ cubic mm. The ruled corpuscles in several groups of 16 squares are counted, and the total, multiplied by 100 (the dilution) and 4000 (representing the size of the cell), and divided by the number of squares counted, gives the corpuscles in a cubic millimetre. In Gowers' instrument the squares are larger and the dilution is greater, but the principle is the same.

The average number of red corpuscles per cubic millimetre is taken to be 5,000,000 for males and 4,500,000 for females. Any greater or less number can be stated as such, or represented as a percentage of the normal. Thus, for males, 3,100,000 corpuscles = 62 per cent.

The number of leucocytes is from 7000 to 10,000 per cubic millimetre. They require to be separately counted, and are usually stated in absolute numbers. In the Thoma-Zeiss apparatus a second pipette provides for a dilution of 1 in 10 with weak acetic acid, by which the red corpuscles are rendered invisible: in Gowers' apparatus the addition of methylene blue to the diluent will help to distinguish the leucocytes.

Estimation of Hæmoglobin.—For this purpose four forms of *hæmoglobinometer* are available, devised respectively by Gowers, Haldane, Fleischl, and Oliver. In Gowers' hæmoglobinometer two tubes are provided, one of which contains gelatine tinted with picrocarminate of ammonia to act as a standard. The other is graduated to 100 degrees, and in this a measured quantity of blood is diluted till it matches the colour of the standard. The figure on the scale which the solution then reaches represents the percentage amount of hæmoglobin. In Haldane's modification of Gowers' instrument, the standard is a 1 per cent. solution of normal blood saturated with carbonic oxide and hermetically sealed in a glass tube. The blood to be tested, diluted with water, and placed in a similar glass tube, is also treated with carbonic oxide or coal-gas; and the comparison is made as before. In Fleischl's apparatus the diluted blood in a glass-bottomed cell is compared with the increasing thickness of a wedge of coloured glass used as a standard; and in Oliver's with a series of tinted glass discs.

Colour-Index.—The percentage of hæmoglobin may be less than that of the corpuscles, if individual corpuscles are smaller, or contain less pigment than normal; the hæmoglobin percentage is greater if the corpuscles are larger, or contain more pigment than normal. This relation of the hæmoglobin percentage to the corpuscle percentage is called the *colour-index*, and it may be less or greater than the normal, 1. Thus, with hæmoglobin, 40 per cent., corpuscles, 50 per cent., the colour-index is $\frac{4}{5}$ or ·8; with hæmoglobin, 30 per cent., corpuscles 20 per cent., the colour-index is $\frac{3}{2}$ or 1·5.

Microscopic Examination of the Corpuscles.—This can only be done satisfactorily after the corpuscles have been stained. The blood is spread on a glass slide, or cover-glass, in a thin layer, or film, dried by heat or in the air, "fixed" by heat or by immersion for three to five minutes in a mixture of equal parts of ether and absolute alcohol, and stained by reagents such as eosin and hæmatoxylin successively, eosin and methylene blue, Jenner's stain, Ehrlich's triacid stain, Leishman's stain, and others.

The different corpuscles which may be seen in health and disease may be now enumerated.

Red Corpuscles, Erythrocytes, or Xanthocytes.—The normal red corpuscle measuring in diameter 7·5μ; small corpuscles, or *microcytes*, from 2–6μ; large corpuscles, or *megalocytes*, from 8–15μ; misshapen, distorted, often pear-shaped corpuscles, or *poikilocytes;* nucleated red corpuscles (*erythroblasts*), divided according to their size into *normoblasts, microblasts,* and *megaloblasts,* or still larger *gigantoblasts.*

Inequality in the size of the red cells is called *anisocytosis*.

Leucocytes.—In health these present many varieties; they are distinguished by their size, the shape of the nucleus, and the presence or absence of granules in the cytoplasm. The small and large lymphocytes are non-granular cells; on the other hand, the polymorphonuclear leucocytes always present granules. In some such cells the granules stain with acid dyes (*oxyphile* or *eosinophile* cells), in others with basic or neutral stains (*basophile*, *neutrophile*), and in others with both acid and basic dyes (*amphiphile*).

An estimate of the relative numbers of the different kinds of leucocytes in a specimen of blood is often of great value in diagnosis, and is called a *differential count*. The relative numbers in normal blood are as follows:

	Measuring about	Per cent.
Polymorphonuclear cells with neutrophile granules	13·5μ	65—75
The same with large eosinophile granules		2—4
Small lymphocytes	5—18μ	28—30
Large lymphocytes	13—15μ	3—6

A distinction is drawn by some writers between large lymphocytes, and *large mononuclear* (or *hyaline*) cells, which are also of large size, with a relatively small nucleus and much cytoplasm.

Transitional cells are found occasionally in healthy blood, but more often in morbid conditions, such as leuchæmia. They are large cells with a much indented or horseshoe nucleus, not as yet broken up like the multiform nucleus of the ordinary leucocyte. The cytoplasm is clear, or may show a few neutrophile granules. They are usually classed with non-granular cells.

Myelocytes or *marrow-cells* occur normally in the red marrow of bones; but they are found in the blood only in diseased conditions and in infancy. The myelocyte is of large size, 20μ in diameter, with neutrophile granules, and a large nucleus, staining very feebly. Myelocytes with eosinophile granules and basophile granules also occur.

Mast-cells are large leucocytes 20μ in diameter, containing coarse granules which stain with basic dyes. Their occurrence in normal blood is not admitted by all writers.

Blood-platelets are round or oval bodies, found in normal blood. They are 2 to 3·5μ in diameter, faint yellow and granular in appearance, adhesive, and thus readily clinging to blood corpuscles and to one another.

Degeneration of the red blood-corpuscles is shown by their colour becoming fainter from the centre outwards, or by irregular appearances of vacuolation, or by irregular points and knobs forming on the corpuscle, which gets deformed and so forms a poikilocyte; and by the protoplasm taking up other stains besides eosin—*e.g.*, hæmatoxylin, when, instead of being pink, it is irregularly violet or blue (*polychromatophile*). Attempts at the *regeneration* of the blood are believed to be indicated by the presence of nucleated red blood cells, especially normoblasts.

Specific Gravity.—This can be ascertained by the method of

Lloyd Jones. A number of aseptic solutions of glycerine in water are prepared, varying in specific gravity between 1030 and 1075. A small quantity of blood is drawn up a bent capillary tube, and is then directed into the centre of different specimens of glycerine solution, until the one is found in which the blood neither sinks nor rises. The specific gravity of this being known, gives, of course, that of the blood.

Coagulation-Period.—Blood from the finger is drawn into a number of glass tubes of standard calibre, which are then kept at a standard temperature, and the time of coagulation is tested by blowing down successive tubes at intervals, and thus finding how soon after withdrawal of the blood coagulation has taken place. (Wright). In health the usual time is from three to six minutes.

Alkalinity.—Wright has also devised a method for estimating the alkalinity of the blood. Blood is drawn from the pricked finger into a small glass bulb or capsule, and allowed to coagulate, and express its serum. By means of a fine capillary tube of drawn glass, measured small quantities of the serum are mixed, each with an equal quantity of twenty-fold, thirty-fold, forty-fold, or intermediate dilutions of pure sulphuric acid, until, as tested with red litmus paper, neutralisation is effected. He finds that the alkalinity of blood in health corresponds to a dilution of about 35, or $\frac{N}{35}$.

Viscosity.—This can be estimated by running a drop of blood from the patient's ear into a specially constructed J-shaped capillary tube, with a bulb in the smaller limb. The time taken by the blood to reach the bulb is noted on a stop-watch, and is compared with the time taken by a drop of water. In health the viscosity of blood ranges between 4·8 and 5·6 times that of water.

Calcium Salts.—These can be estimated by the method of Dr. Blair Bell. A measured quantity (100 c.mm.) of blood is added to 250 c.mm. of solution of oxalic acid (1 in 30) in a specially constructed glass capsule. After standing for ten minutes or more, 250 c.mm. of a 1 per cent. solution of glacial acetic acid are added. The mixture is shaken and again allowed to stand. Then 100 c.mm. are mixed with 500 c.mm. of distilled water, a drop is placed on the hæmocytometer plate, and the number of calcium oxalate crystals per square is counted under a magnification of 450 diameters.

Nomenclature.—Recent advances in hæmatology have revealed new conditions which call for an increasing number of complex names. Originally *anæmia* or *spanæmia* signified pallor in the patient, and a thin pale blood as it issued from a wound; and the excess of white corpuscles recognised by Virchow in 1845, and called by him *leukæmia*, has been since called by that name, or by the name *leucocythæmia*, proposed by Hughes Bennett in 1851. When enumeration of corpuscles became possible, less degrees of white-cell increase, both physiological and reactive in disease, were recognised as *leucocytosis*, while their deficiency was called *leucopenia*

(πενία, poverty). Oligocythæmia was used to signify so much of the anæmia as belonged to the red corpuscles independent of hæmoglobin. The last event is the recognition that the red corpuscles, or erythrocytes, may be in enormous excess. This condition was at first called *polycythæmia*, but the term is being replaced by two others, *erythræmia*, and *erythrocytosis*, of which the former denotes a primary excess, the latter an increase in response to special demands. We thus have:

Deficiency of blood in general (corpuscles and hæmoglobin)—anæmia.
„ „ the whole quantity of blood—oligæmia.
„ „ red corpuscles—oligocythæmia, erythropenia.
„ „ hæmoglobin—oligochromæmia.
„ „ white corpuscles—leucopenia.
Excess of blood in general (red corpuscles, hæmoglobin, plasma)—polyhæmia.
„ „ red corpuscles—erythrocythæmia, erythræmia, erythrocytosis, polycythæmia.
„ „ white corpuscles—leuchæmia, leucocythæmia, leucocytosis.
„ „ different forms of leucocytes—lymphæmia, myelæmia, eosinophilia.

It will be observed that all pronounced forms of leuchæmia are accompanied by anæmia, or, more strictly, erythropenia: and, further, that both absolute and relative increase and decrease of particular kinds of corpuscles may have to be estimated.

ANÆMIA

The normal pink colour of the skin and the deeper red colour of the mucous membranes are due to the blood circulating in their vessels: if a considerable loss of blood occurs—as, for instance, after injury, or from gastric ulcer, or during parturition—the natural colour is materially altered, and the skin becomes of a waxy whiteness, blanched, or bloodless, and even the lips and mucous membranes have only a very pale pink colour. This condition is spoken of as *anæmia*, and occurs in a great variety of circumstances, besides the direct loss of blood in quantity; moreover, it is accompanied in all its forms by numerous other disturbances, which directly result from the deficiencies which exist in the blood itself.

Causation.—The distinction commonly made between primary anæmia and secondary or symptomatic anæmia is obviously unsatisfactory. In so-called secondary anæmia, there is an apparent cause. The bloodlessness results either from direct losses of blood from the system, or from more or less definite diseases of particular organs, whereby the income or expenditure of the body is materially affected. In so-called primary anæmia the change seems to depend on the blood itself; but the high probability is that such cases are due either to toxins operating upon the blood-forming organs,

including the bone-marrow, or to *hæmolytic* agencies destroying the blood-corpuscles.

The two diseases which have been regarded as typical instances of a *primary* anæmia are *chlorosis* or *green sickness*, which occurs so frequently in females about the time of puberty; and *idiopathic* or *pernicious anæmia*, which occurs in adults of both sexes: but one or both of these may be septic or toxic in origin, and hence become related to some anæmias of the next group.

The anæmia called *secondary* or *symptomatic* includes that due to (1) hæmorrhages of all kinds; this may be from incised or lacerated wounds, from epistaxis, gastric, intestinal (typhoid), or rectal ulceration, piles, excessive menstruation, uterine fibroid, ulcerating cancerous tumours, and other lesions. Many of these hæmorrhages are profuse, but do not recur, or only at long intervals; the anæmia is in direct proportion to the loss, and recovers perhaps quickly within a short time. Repeated small hæmorrhages may take place from piles, rectal ulcer, and uterine diseases, in which case the anæmia is persistent. Another form of hæmorrhage is that due to a diseased condition of the blood itself, such as occurs in purpura, scurvy, malarial poisoning, jaundice, and Bright's disease; but in some of these the anæmia is in part due to the condition of the blood which causes the hæmorrhage, and may be present before bleeding takes place. Anæmia is also caused by (2) destruction of blood-corpuscles in the circulating blood, as by the malarial parasite and in hæmoglobinuria; by (3) continued discharges of pus from old sinuses, or leucorrhœal or other discharges in women; by (4) deficient ingestion of food, as in starvation or stricture of the œsophagus; and by (5) deficient assimilation, as in cancer of the stomach, of the liver, and of other assimilating organs. Assimilation is defective in the course of severe acute illnesses, such as enteric fever, pneumonia, pleurisy, and rheumatic fever; and pallor is a constant accompaniment of the later stages of this illness and its early convalescence. Indeed, most chronic diseases affecting the viscera sufficiently interfere with blood-formation to cause anæmia—*e.g.*, phthisis, Bright's disease, aortic regurgitation, and all kinds of valvular disease in children. (6) Some other diseases affect the blood in a way that is not fully understood, and produce anæmia, such as lead-poisoning and syphilis. (7) The intestinal worms, bothriocephalus latus, and ankylostoma duodenale, cause anæmias, of different types, and in neither case the result of abstraction of blood by the worm. (8) Diminution of the red corpuscles occurs in Hodgkin's disease, and in the different varieties of leuchæmia. These are primary diseases of the spleen, lymph-glands, and bone-marrow, or other blood-forming organs.

The above enumeration shows that some forms of anæmia are caused by a great loss of corpuscles (hæmorrhage), or by excessive destruction of them (malaria), and others by their defective formation (starvation, chronic diseases, diseases of blood-forming organs). Destruction of the blood is called *hæmolysis*, its formation *hæmo-*

genesis; and anæmia may result in any case in which the balance of these two processes fails to be maintained, either from excess of hæmolysis or defect of hæmogenesis.

There is much yet to be learned as to the exact cause in particular cases either of undue hæmolysis or of defective hæmogenesis, and of the influence of toxins or other poisons upon the blood-forming organs, especially the bone-marrow.

Symptoms of Anæmia.—Some features are common to all cases of pronounced anæmia, however caused. The skin is pale and waxy-looking. In cases of recent hæmorrhage the colour is almost white, but in pernicious anæmia there is a yellow tinge; in chlorosis a greenish tint is sometimes detectable, and a dirty yellow, earthy, or sallow tint in the anæmia of lead-poisoning, malaria, and syphilis. The lips are pale pink, and the cheeks may show a faint pink flush. The visible mucous membranes are pale pink, as seen in the mouth, the tongue, and the inner side of the eyelid. The altered colour of the blood is also manifest in the tint of the veins on the back of the hand, which show pink through the white skin, instead of dark purple through the pink skin. The patient is languid and weak, unfit for physical or mental exertion, and liable to headache, vertigo, the appearance of spots before the eyes, ringing in the ears, and attacks of syncope. In sudden and large losses of blood, such as occur in parturition, the anæmic condition of the brain may produce convulsions, but these do not occur in chronic cases, unless it may be near the fatal termination. There is dyspnœa on exertion, palpitation of the heart, and throbbing of the vessels; in some cases œdema of the feet may be present. The appetite is generally diminished, and the ingestion of food is often followed by weight or oppression at the epigastrium, or by severe cardialgia.

Any marked degree of anæmia is accompanied by murmurs over one or more of the cardiac orifices. The most common is a systolic murmur, often harsh in quality, heard loudest in the second left intercostal space, and traceable outwards along that space and towards the left clavicle—that is to say, in the area of the pulmonary artery. This murmur is often loudest in the recumbent position, and diminishes or even disappears when the patient stands up. In some cases a murmur is heard at the apex, and in more severe anæmia, systolic murmurs may be heard at all the orifices, or even the whole cardiac area, as well as behind. A similar murmur is often heard in the carotid vessels in the neck. A murmur is also commonly heard at the root of the neck over the jugular vein, the *bruit de diable,* which has already been described (*see* p. 578). Neither this nor the systolic pulmonary hæmic murmur is peculiar to anæmia, nor should either be regarded as evidence of it; but they are almost constantly present in this condition, and are generally more marked in proportion to its intensity.

The pulmonary hæmic murmur probably depends less on the quality of the blood than on the changes which anæmia produces

in the myocardium and the arterial walls. Attention has long been directed to the dilatation of the conus arteriosus as shown by the visible pulsation in the second left intercostal space, and to the dilatation of the base of the pulmonary artery seen *post mortem ;* and this enlargement of the ventricle is believed to force up and distort the pulmonary artery, producing an aneurysmal sacculation of its anterior wall. The increased loudness of the murmur in the recumbent position is compatible with this explanation.

The dilatation of the heart already mentioned carries the impulse into the line of the nipple, or even external to it. The pulse is commonly rather quicker than normal; generally soft, and rather full, but sometimes hard in slight cases; and the heart's action is readily quickened by exertion or excitement.

In severe degrees of anæmia hæmorrhages are liable to occur, and the temperature may be raised. Retinal hæmorrhages and pyrexia are, indeed, common in pernicious anæmia, but they are also seen in chlorosis and other forms occasionally. Another occasional result of chlorotic anæmia is venous thrombosis.

The **Anatomical Changes** associated with anæmia are illustrated by cases of pernicious anæmia, but the possible influence of toxins, as well as anæmia, must not be ignored (*see* p. 834). The organs are universally bloodless, and in some cases of secondary anæmia is found the fatty degeneration of the muscular fibres of the heart (tabby-cat striation) which is constant in the pernicious forms. It appears to be due to an imperfect process of oxygenation, which is the result of a less quantity of hæmoglobin.

CHLOROSIS

This name—or its English equivalent, green sickness—is applied to a form of anæmia which especially occurs in girls and young women between the ages of fourteen and twenty-four, though a similar condition is exceptionally seen in boys. In its milder varieties it presents the characteristics already described; in its severer forms it may be difficult to distinguish from pernicious or Addison's anæmia. Languor, feebleness, dyspnœa, and palpitation on exertion, vertigo, syncope, headache, noises in the ears, spots before the eyes, nausea, eructations, pain after food, and constipation, occur just as in cases of secondary anæmia. The patient may present all degrees of pallor, often with some temporary flushing of the cheek or lips; sometimes also, no doubt, there is a tint which justifies the name *chlorosis,* but it can scarcely be recognised in all cases—Zimmermann says it occurs only in those of a dark complexion. Both blood-corpuscles and hæmoglobin as tested in the usual way are diminished; the former are rarely below 60 per cent. of the normal, but the hæmoglobin may be only 25 or 30 per cent. Thus each corpuscle has on an average less than its normal amount of hæmoglobin—that is, the colour-index is less than unity. This relation of corpuscles to hæmoglobin is often called the *chlorotic type ;* but it occurs also in

other forms of anæmia. Microcytes and poikilocytes may be found in small quantities, but in severer cases the latter may be very numerous: normoblasts appear occasionally. The leucocytes are normal, or there may be a slight excess of lymphocytes. The diminution of the corpuscles and hæmoglobin is probably not nearly so great as it appears, for examinations of the volume and total oxygen capacity of the blood (Haldane and Lorrain-Smith) show that the volume is much in excess of the normal, and that there is thus a great dilution of the blood, or a condition of *hydræmic plethora*, which will in great part explain the symptoms. The specific gravity is low: and coagulation is rapid. There are murmurs at the base of the heart and over the jugular veins. The pulse is not necessarily feeble; its tension may be, but it is not always, increased. An almost constant symptom, which more than all attracts the attention of the patient or her friends, is the suppression or diminution of the menstrual flow. In the younger patients the menses may never have appeared, and may continue absent as long as the chlorosis lasts; in those who have already menstruated, the flow becomes scanty, pale, or irregular, or ceases altogether. Only occasionally are the menses more abundant than normal. It is possibly in connection with these menstrual disorders that the mental condition of the patient is often markedly affected, as shown in irritability and a tendency to hysterical manifestations. Constipation is frequent, and the urine is pale. Optic neuritis occasionally occurs, and may be followed by atrophy: exceptionally also ocular paralysis, retinal embolism, and retrobulbar neuritis. All these may be attributable to the occurrence in a limited area of intracranial thrombosis (Hawthorne). A more extensive thrombosis is well known to cause profound cerebral symptoms, with a fatal result (*see* p. 363).

Pathology.—Numerous hypotheses have been put forward to explain the occurrence of chlorosis. The following are some of them: (1) Disorders of menstruation, especially amenorrhœa; (2) Congenital narrowness of the aorta, which was found pathologically by Virchow, and is referred now to a hypoplasia of the mesoblastic layer; (3) Disorders of the stomach, amongst which are (*a*) excessive acidity of the gastric secretions, (*b*) proptosis of the stomach, (*c*) gastric ulcer and hæmorrhage. Tight-lacing has been considered a possible cause of proptosis and hæmorrhage; (4) The presence in the blood of unassimilable forms of iron, from deficiency of hydrochloric acid in the system (Zander); (5) Sepsis from the alimentary canal, whether oral (*pyorrhœa alveolaris*), gastric, or intestinal (habitual *constipation*). Bunge thought that the organic iron compounds of the food (*hæmatogen*) were broken up by decomposition in retained fæces, and assumed forms less capable of being absorbed. Undoubtedly there must be some connection between the sex, puberty and the onset of chlorosis; but amenorrhœa is a consequence and not a cause. Whether defective hæmogenesis or hydræmic plethora is the chief fault in the blood, there is no adequate explanation of its occurrence as yet forthcoming.

Diagnosis.—The recognition of chlorosis is generally easy; but it may be difficult, in some severe cases where the heart is dilated and murmurs of mitral regurgitation are heard, to decide whether the cardiac lesion is primary, or a result of the anæmia. Amenorrhœa, the absence of rheumatism, scarlatina, or chorea in the history, and extensive præcordial murmurs, rather than one localised to the heart's impulse and the back, would be in favour of chlorosis. The presence of any primary lesion (*see* p. 828) must always be carefully excluded.

Prognosis.—Chlorosis is rarely fatal; it often lasts months or years, and frequently recurs after cure.

Treatment.—Any of the above conditions which may seem to be in operation should, if possible, be dealt with. But for the restoration of the blood-state in chlorosis, the use of iron is of the first importance. This may be given in several forms: if there is irritability of the stomach, the less astringent forms are advisable, such as reduced iron, 2 or 3 grains three times a day, the ammonio-citrate, or the tartrate, 5 to 10 grains. The more astringent forms, however, when they can be borne, are more quickly efficient—*e.g.*, the perchloride, 10 to 20 minims of the tincture, or the sulphate, from 3 to 5 grains. A well-known remedy is Blaud's pill, containing $2\frac{1}{2}$ grains of sulphate of iron and the same of potassium carbonate. One, two, or three pills are given thrice daily. The Pilula Ferri, B.P., is a similar combination. Iron should always be given immediately after meals. It is always desirable to combine some laxatives with iron, both to counteract its astringency as well as to overcome the tendency to constipation, which is so frequent. This may be done by the use of sulphate of magnesium, in combination with the perchloride or the sulphate of iron; or by the use of aloes, in the form of aloes and iron pills; or an aloes or myrrh pill may be given at night with the iron mixture. In some cases arsenic is of value in connection with iron. The diet must be good and nourishing, and may be modified somewhat according to the fat or lean condition of the patient. In severe or advanced cases, treatment by drugs may fail entirely until the patient has had a prolonged rest or three or four weeks on the couch, or even in bed. Exercise increases both the tendency to dilatation of the heart and the overstrain upon the blood-forming organs; and, though fresh air is desirable, it should be obtained without exertion on the part of the patient.

PERNICIOUS ANÆMIA

(*Addison's Anæmia*)

Cases of this disease were first described by Addison under the name of *idiopathic anæmia*, because they presented distinctive features, and he was unable to find a cause for them. Later, Biermer and other Continental writers described similar cases, under the name of *progressive pernicious anæmia:* and no doubt included

cases arising secondarily from definite causes, such as syphilis, cancer, and repeated hæmorrhages.

Ætiology.—It affects both sexes equally, and is most frequently seen between the ages of twenty-five and forty. Its origin is often quite obscure; pregnancy and the puerperal state have been credited with its causation in many cases, and other antecedents are inflammatory lesions about the mouth and tongue, oral sepsis and pyorrhœa alveolaris, gastro-intestinal disturbance, cancer, syphilis, privation, mental shock, and large hæmorrhages occurring at long intervals before the anæmia.

Symptoms.—The patient with pernicious anæmia gradually loses strength and becomes paler; his skin acquires a yellow tint, different from the waxy white of ordinary anæmia, but it is a lemon-yellow tint rather than the greenish-yellow of bile-pigment. Sometimes there is brown pigmentation in small or large patches. At the same time, even with extreme anæmia, the patient does not lose flesh, and the subcutaneous fat may be abundant. There are the same languor, indisposition for mental and physical exertion, giddiness, noises in the ears, &c., which have been described in the other forms, as well as dyspnœa on exertion, cardiac palpitation, and vascular murmurs. The patient complains of dryness of the mouth and throat, with soreness of the tongue, which presents pimples, abrasions, and even ulcers. Loss of appetite and nausea are frequent; and in a large proportion of cases there are vomiting and diarrhœa. The urine is usually high-coloured from excess of urobilin, and is free from albumin. It may contain a full quantity of urea and uric acid, as well as indican. The bones are often tender on percussion or pressure. The pupils are mostly dilated; and the retina shows numerous small hæmorrhages, which are abundant round the optic disc. These are striated, or flame-shaped, and may be accompanied with white spots. Another feature of idiopathic anæmia is the presence of fever, which may give a temperature of 101° or 102°, but is generally irregular. It may be absent for days together, and the temperature is often subnormal before death. Sometimes the liver is enlarged and tender: occasionally the spleen is large. The blood is excessively pale, and the red corpuscles are reduced to 2,500,000 per cubic mm., or even to 500,000 or lower; but the diminution of the hæmoglobin is less in proportion. Thus the colour-index is greater than 1, and may be even 2. This contrasts with the condition in chlorosis, and is due to the fact that individual corpuscles are relatively large and contain excess of pigment. Megalocytes are common, and poikilocytes are more numerous than in any other condition. Usually there are some nucleated red cells, especially megaloblasts, but the leucocytes are commonly less numerous than normal, with a relative lymphocytosis. The specific gravity and alkalinity are diminished; and there is a marked diminution of coagulability.

The course of the disease is not always progressive: indeed, remissions are frequent, especially under treatment: and the

patients regain colour, and the condition of the blood is much improved. Ultimately, however, in most cases they gradually get weaker, become drowsy and apathetic, and death takes place.

Exceptionally, patients with pernicious anæmia have numbness and weakness of the legs, ataxy, altered knee-jerks, and other symptoms of spinal sclerosis (*see* p. 289).

Anatomical Changes.—Besides the universal pallor of the organs, one of the most constant conditions *post mortem* is fatty degeneration of the heart, principally in the form of the striation already mentioned. It occurs in the left ventricle and on the papillary muscles. There is also fatty degeneration of the liver and kidneys, and of the intima of the arteries. Hæmorrhages are found not only in the retina, where they have been seen during life, but in the serous membranes, the endocardium, the mucous membrane of the stomach, the lungs, the surface of the brain, and other parts. Fenwick and others have found fatty degeneration or atrophy of the tubular glands of the stomach, and infiltration of leucocytes between the tubules. The spleen is sometimes enlarged, and of dark red or purple colour. The marrow of the bones has been found to be of a reddish-purple colour, with large numbers of nucleated red corpuscles, especially megaloblasts, while the fat cells are all or nearly all destroyed—a return to the condition of the embryo. There is, moreover, an abundant deposit of iron in the cells of the liver and in the spleen, as can be shown by the organ turning black with ammonium sulphide, or blue with potassium ferrocyanide and dilute hydrochloric acid. Whereas the normal iron contents are ·1 per cent., in this disease they may rise to ·7 per cent. In the liver the iron is most abundant in the peripheral zones of the lobules. In cases with spinal symptoms, a combined lateral and posterior sclerosis is found after death.

Pathology.—Different opinions are still held with regard to the pathology of this complaint. Probably the changes in the bone-marrow are secondary, and the general belief is that the disease is toxic in its nature.

The striking feature is the evidence of blood-destruction (hæmolysis) in the excess of urobilin in the urine, the deposit of iron in the liver, and the presence of megaloblasts in the blood, showing efforts at regeneration in the bone-marrow. This hæmolysis probably takes place within the portal circulation. The alimentary canal has been looked to as the source of infection, as indicated by vomiting and diarrhœa in some cases, by septic changes about the teeth and gums in others. Hunter is convinced that it is an infective disease, shown especially by a special inflammation of the tongue (infective glossitis), and he would include as instances of the disease (Addisonian anæmia) only the cases in which hæmolysis was shown by excessive urobilinuria, and increased iron deposit in the liver, and in which the colour-index was excessive—*i.e.*, more than unity. It is interesting that the anæmia due to bothriocephalus

latus resembles it in the diminished red cells and hæmoglobin, the high colour-index, and the presence of megaloblasts (*see* p. 758).

Diagnosis.—In any supposed case of *Addisonian* or *pernicious anæmia* it is important to search most carefully for organic disease, such as cancer, which might be the cause of the bloodlessness, and for septic foci; and in certain circumstances to exclude intestinal worms (bothriocephalus) by proper examination of the fæces. The distinctive features of the disease are the lemon-yellow tint of the skin, the retinal hæmorrhages, the absence of wasting, the great number of poikilocytes in the blood, the presence of megaloblasts, and especially the great diminution of red corpuscles and the high colour-index. The latter with the dark colour of the urine serve to distinguish it from chlorosis and from ankylostoma anæmia (*see* p. 761).

Prognosis.—This is very unfavourable, but temporary recovery is often seen under the use of arsenic, with relapse after some months. Many cases get progressively worse and die in about six months; whereas others last from twelve months to two years.

Treatment.—Arsenic seems to be the most efficient remedy of all drugs that have been tried; and it should be pushed to full doses. Fresh bone-marrow of the ox or calf (3 ounces daily) has been successful in some cases. If the septic theory of the disease be accepted, a thorough antiseptic treatment should be pursued. The teeth should be seen to, the mouth should be rinsed with antiseptic lotions (formalin, hydrogen peroxide), and creosote, salol, or salicylate of bismuth may be given internally. Antistreptococcus serum has been injected subcutaneously. In extreme conditions the end may be postponed by transfusion of a saline solution.

ALLIED FORMS OF SEVERE ANÆMIA

Hunter distinguishes from Addison's anæmia a *septic anæmia*, also arising from oral, gastric, or intestinal sepsis; but in this the colour-index of the corpuscles is not very different from unity; the evidences of hæmolysis (lemon-yellow skin and urobilinuria) are absent, and the prognosis is relatively favourable. Oral sepsis may be present with the glossitis in Addison's anæmia, and materially delays the success of treatment by arsenic.

An *aplastic anæmia* has been described which runs a fatal course. The red corpuscles and hæmoglobin are reduced to 20 per cent. of the normal, and the colour-index is below unity: there is leucopenia with relative lymphocytosis, and an absence of normoblasts and megaloblasts. The bone-marrow differs from that of pernicious anæmia in being remarkably pale and fatty, and in wanting all signs of blood-regeneration. Thus there is *aplasia* of the bone-marrow, which it is suggested may be due to inhibition by some toxin. Since, in some cases, there have been antecedent severe hæmorrhages (metrorrhagia, epistaxis), the

condition of the bone-marrow has been also attributed to an excessive demand upon its blood-forming function.

SPLENIC ANÆMIA

This name is given to a condition in which great enlargement of the spleen is associated with profound anæmia. The first event is either a general anæmia or an attack of hæmatemesis, or some complaint of pain in the left side, probably due to attacks of perisplenitis. When first observed the spleen has often reached a great size, and in the course of the illness it may be large enough to extend forwards to the umbilicus and downwards to the iliac crest. The anæmia is considerable, of chlorotic type, the red corpuscles ranging from 2,000,000 to 3,000,000; and the hæmoglobin from 35 to 50 per cent. The leucocytes are generally in less number than normal (leucopenia), that is, often 4000 or 5000 per cubic mm. Normoblasts and a few megaloblasts may be present.

The disease runs a long course, often three or four years, sometimes ten or twelve years, and the anæmia slowly increases. The hæmatemesis may be repeated, and other hæmorrhages may occur, such as epistaxis or retinal hæmorrhage. The liver is also slightly enlarged, and there are digestive troubles: but there is no enlargement of the lymphatic glands. In some cases there is marked pigmentation of the skin. It occurs in both sexes, and at all ages from childhood up to late middle age; and instances of its occurrence in two or more members of the same family are on record. In some cases after a long time the liver becomes still more enlarged, and definitely cirrhotic. Ascites then follows, though it also sometimes occurs without cirrhosis. The addition of definite cirrhosis of the liver to splenic anæmia constitutes *Banti's disease.*

Pathology.—The spleen is found to be greatly enlarged, weighing from two to four pounds, firm and generally smooth, presenting hæmorrhages and infarcts on section. Under the microscope is found a great increase of the fibrous trabeculæ and septa, atrophy and fibrous transformation of the Malpighian corpuscles, and great numbers of large endothelial cells containing numerous nuclei and blood-corpuscles. The bone-marrow has been found in a few cases to be red or purple in colour, or to present circumscribed lymphoid or lymphadenoid spots; in one case it presented gelatinous degeneration with no signs of regeneration.

The sequence of events is by no means clear as yet. That the spleen has some influence in the production of the anæmia is shown by the fact that splenectomy has in several cases been followed by cure, the blood-corpuscles more or less rapidly reaching the normal amount. The assumption from this fact is that the anæmia is due to a morbid increase of the function of the spleen, namely, the destruction of the blood-corpuscles by its endothelial cells. But there is no excess of iron

in the liver, such as is seen in pernicious anæmia: and the bone-marrow is diseased in some cases. The hypothesis of an auto-intoxication remains unproved.

Diagnosis.—The disease may be confounded with myelocytic leuchæmia, with pernicious anæmia, and with infective endocarditis. Its characteristic features are the anæmia of chlorotic type, the leucopenia, the large size of the spleen, the long duration, and the occurrence of hæmorrhages. An examination of the blood at once distinguishes it from leuchæmia: a confusion with malignant endocarditis is possible, because in this last disease the spleen may be very large, the anæmia may be pronounced, and purpura and hæmorrhages may occur; while hæmic murmurs may be present in splenic anæmia.

Treatment.—Iron, arsenic, and other hæmatinics are of little value: and if after a period of observation the diagnosis is established, the removal of the spleen is justified. Although it is not free from the danger of hæmorrhage, cases have been completely successful. In one case, a preliminary examination of the bone-marrow, obtained by trephining, showed that it was diseased, and splenectomy was abandoned. Röntgen rays may be tried, as in leuchæmia (*see* p. 842).

SECONDARY ANÆMIA

The condition of the blood in cases called *secondary anæmia* is somewhat like that of chlorosis. The corpuscles may not be very much diminished in number, but the loss of hæmoglobin is relatively greater. Microcytes as well as poikilocytes occur in the severe cases, and in the worst cases nucleated red cells, especially normoblasts. The leucocytes are variable; they are often increased in number, especially in cases dependent on inflammatory and pyogenic conditions. The coagulation-time is shortened.

LEUCHÆMIA

(*Leucocythæmia*)

In this disease there is a considerable and persistent increase in the number of white corpuscles in the blood, associated with changes in the spleen, the lymphatic glands, or the marrow of the bones.

In all varieties of leuchæmia, the bone-marrow is diseased, in many the spleen, in some the lymphatic glands, and in a few all three of these blood-forming organs: in all varieties the natural proportions of the various leucocytes to one another are profoundly altered, and in some the blood contains myelocytes or marrow-cells, which are not present in it in health.

Until recently leuchæmia was divided into two groups, splenomedullary and lymphatic, on the hypothesis that in one group the

spleen and the medulla of the bones were primarily diseased, and in the other the lymph-glands: and different conditions of the blood were believed to characterise the two groups. It is now recognised that no such simple division exists; that in association with a blood-count supposed to characterise lymph-gland enlargement, the spleen may be greatly enlarged, and the lymph-glands may be small; that the blood may present mixed types of change; and further that the cells characteristic of one so-called type may be in a short time replaced by the cells characteristic of the other.

It is convenient to base divisions of leuchæmia upon the condition of the blood, and the predominance of this or that form of leucocyte.

The two chief varieties which are known to occur are (1) one in which myelocytes are found in the blood to the extent of 30 or 40 per cent. of the leucocytes; this is *myelocytic leuchæmia*, or *myelocythæmia*, and corresponds to the cases formerly called *splenomedullary*. (2) Another in which lymphocytes are in the blood to the extent of 90 or 95 per cent. of the leucocytes; this is *lymphocytic leuchæmia*, or *lymphocythæmia*, and corresponds to many cases called *lymphatic*. In some of these cases, the lymphocytes are almost entirely *large lymphocytes;* in others almost entirely *small lymphocytes*.

But some minor details should be noted. One is that in all cases of leuchæmia some large lymphocytes are present: and that in cases of lymphocythæmia there are nearly always a few myelocytes. Transitions from small lymphocytic to large lymphocytic leuchæmia, and from myelocytic to large lymphocytic leuchæmia—that is, the replacement of small lymphocytes by large lymphocytes and of myelocytes and polymorphonuclears by large lymphocytes—have been observed. But in no case so far have small lymphocytes been replaced by myelocytes.

The red corpuscles are always diminished in number, and the hæmoglobin also, with a colour-index less than unity. Another variety of case closely allied to leuchæmia is the form which has been called *leucanæmia:* in this the blood presents the characters of pernicious anæmia, so far as the erythrocytes are concerned, combined with a leuchæmia either of myelocytic or of lymphocytic type. The organs are affected like those in leuchæmia.

In all these deviations, whether mixed or transitional, from the more common type, the symptoms and course of the disease are not materially altered.

Ætiology.—The cause of leuchæmia in any form is really unknown, and there are no constant antecedents to explain its occurrence. The myelocytic variety occurs in men more often than in women, and mostly in middle life; though sometimes in quite young children, but rarely in infants. Lymphocytic leuchæmia is more common in young people.

Pathology.—Little is really known of the origin of leuchæmia. Apparently in all cases the bone-marrow undergoes some form of irritation, by toxins or otherwise, and produces an excess of

leucocytes with a less formation of red corpuscles or erythrocytes. It is believed that the original parent-cell of the marrow is a non-granular, nucleated corpuscle, from which (1) the red cells are developed; (2) the non-granular leucocytes, the large lymphocyte preceding the small lymphocyte; and (3) the granular leucocytes, the myelocyte developing into the polymorphonuclear leucocyte. In the very acute cases the predominant cell is the large lymphocyte, which is the most closely allied to the original marrow-cell. The suggestion is obvious that in the hasty process this cell has not had time to undergo any further development; whereas in a chronic lymphocytic case the predominance of the smaller, older cell suggests that more time has been allowed. In the myelocytic case also more than half the leucocytes are in the undeveloped myelocyte stage, and only the remainder of the granular cells are the fully developed polymorphonuclears. In the transitions also we may suppose that the marrow is pushing forward into the blood the early, large lymphocytes, which have had no time to develop either into the small lymphocyte in the one case, or into myelocyte and polymorphonuclear in the other. That the small lymphocyte does not in the same case get replaced by myelocyte is because they are not genetically related to one another, as they both are to the large lymphocyte. What is the share of the spleen and lymph-glands in the production of the leucocyte excess is very uncertain; their enlargement when it occurs may be due to their diseased activity, or it may be, as some think, simply due to the organ being densely packed with the leucocytes circulating in excess in the blood.

MYELOCYTIC LEUCHÆMIA

(*Myelocythæmia, Myeloid Leuchæmia*)

Condition of the Blood.—In well-marked examples of leuchæmia the blood is pale and thin as it issues from a wound, and as seen after death it is grumous-looking, or forms pale pus-like clots; its coagulation, moreover, is imperfect from the fibrin being deposited in a granular, rather than in a fibrous, form. Under the microscope the white corpuscles are seen to occupy nearly the whole of the field, instead of being few and scattered as in health; on the other hand, the red corpuscles are less numerous than in health. The white corpuscles are found to number from 200,000 to 900,000 in the cubic mm., instead of 7000 or 8000; and the red corpuscles may be from 3,000,000 to 2,000,000, or even as low as 1,000,000. The hæmoglobin is less in proportion than the red corpuscles, *i.e.*, the colour-index is less than 1, and the blood presents the chlorotic type. When the leucocytes are more fully examined, it is found that from 30 to 50 per cent. of them are *myelocytes* or marrow-cells: the *polymorphonuclear cells* are from 40 to 50 per cent., that is, relatively to other leucocytes less, though actually more than in health; *eosinophiles* are increased in the same proportion as

the other leucocytes, forming from 2 to 4 per cent.; while the *lymphocytes* are relatively diminished, forming from 4 to 7 per cent.

Poikilocytes are present, nucleated red cells (normoblasts and megaloblasts) are numerous, and mast-cells are often found. Charcot-Leyden crystals (*see* p. 495) have been found in the blood, and in the spleen and other organs after death. Chemical examination shows a great increase of the xanthin-bases, which are believed to result from the destruction of the leucocytes. Lactic, formic, and mucinic acids have also been found.

Symptoms.—One of the first indications of leuchæmia, in a great number of cases, is the swelling of the abdomen from the *enlargement of the spleen*, which may have been developing for some time without giving any sign. It may then be found occupying the whole of the left side of the abdomen, forming a firm, hard tumour, which extends backwards into the flank, while its anterior margin begins about the ninth costal cartilage, reaches the middle line at the umbilicus level, and not infrequently below this extends two or three inches to the right. This position is determined by its attachment to its vessels, which compel it to enlarge along the circumference of a circle of which the cœliac axis is a centre. The anterior margin is more or less sharp, and presents one or two notches. In earlier stages the spleen only occupies the left hypochondriac region, like the enlargements in ague, and in some cases of typhoid fever. The *liver* is moderately enlarged, and can be felt for one or two inches below the right costal margin. The implication of the *bone-marrow* is sometimes shown by tenderness on pressure or percussion of the corresponding bone.

The general effects of the illness do not always show themselves early; especially it must be observed that when the spleen is already very large, and the leuchæmia unmistakable, there may be no pallor of the skin, lips, and mucous membranes. Later on, however, the patient loses colour, and becomes sallow, or markedly anæmic.

The *temperature* is generally affected in this disease; there is either continuous moderate pyrexia, or there are periods of pyrexia alternating with periods of apyrexia; and the febrile reaction sometimes gives a flush to the skin, which may help to mask the approaching anæmia.

The altered condition of the blood shows itself in the occurrence of *dyspnœa* and of *hæmorrhages*, which last chiefly take the form of epistaxis, bleeding from the gums and mouth, and purpuric spots under the skin; but also occasionally bleeding from the lungs, stomach and intestines, kidneys, or uterus, or hæmorrhage into the brain. Hæmorrhages also occur in the retina, where they may be seen with the ophthalmoscope associated with white streaks and spots, said to consist of masses of leucocytes; and the retinal veins are often remarkably tortuous (*leuchæmic retinitis*). Other organs may be affected, but the changes are chiefly observed after death. The urine is acid, of high specific gravity, uric acid and xanthin-

bases are in excess, and indican is often present. Albumin is rare unless the kidneys are diseased.

The course of the disease is generally progressive until its termination in death, and it lasts from six months to four years. Towards the end the pallor increases, the feet and other parts of the body became œdematous, ascites and hydrothorax may be added, the pulse is quickened, and palpitation is frequent. Diarrhœa is occasionally a prominent symptom. Finally, death takes place from loss of blood, asthenia, diarrhœa, pleurisy, pneumonia, bronchitis, or cardiac dilatation; and occasionally from cerebral hæmorrhage.

Anatomical Changes.—The *spleen* often weighs five or six pounds, but a weight of eighteen pounds has been recorded. It is uniformly enlarged, and presents its normal shape; on the surface are often patches of thickening of the capsule, and the organ is more or less adherent to the abdominal wall, diaphragm, or adjacent viscera. On section it often has a brownish, rather than a red colour, homogeneous, or marked with paler lines due to thickened trabeculæ. It is smooth, hard, and dry. Not infrequently there are large wedge-shaped infarcts, either yellow and caseous or red and hæmorrhagic. The change in the spleen itself is one of great increase of the splenic pulp, which is full of the same cells as are found in the blood, and the outlines of the Malpighian bodies are badly defined: in long-standing cases the stroma becomes more fibrous, and the trabeculæ are increased in thickness.

The *liver* is enlarged, and may reach twice or three times its normal size. It is pale and smooth, and presents under the microscope a dense infiltration with leucocytes, which for the most part surround the portal vessels in their distribution, but are partly in nodular masses. The vessels also are full of leucocytes. The *kidneys* are pale, and enlarged from granular degeneration of the cells, and distension from leucocytes; or they present grayish-white deposits, running like striæ between the cortical tubules. There may be stomatitis, or pharyngitis, swelling of the *tonsils*, and of the *follicles* at the root of the tongue; and swelling and superficial ulceration of the follicles of the intestine. The *thymus*, *thyroid*, and *suprarenal bodies* may also be diseased, and tumours in the skin have been recorded. Sometimes the *lungs* present hæmorrhagic infarcts. The *marrow* of the bones is either yellow and pus-like, or pink and firm, the fat of the marrow being replaced by a tissue like that of active marrow, in which marrow-cells and nucleated red cells are abundant. Besides the occasional hæmorrhage into the brain, diffuse sclerotic changes and scattered areas of acute inflammation have been found in the *brain* and *spinal cord*.

Diagnosis.—The spleen is easily recognised by its position on the left side, extending from the ribs to the pelvis, with its irregular anterior margin; but no diagnosis can be safely made unless the blood is examined. An equally large spleen may be seen in *lymphocytic leuchæmia*, in *splenic anæmia*, in *polycythæmia*, and in *heart disease*, especially malignant endocarditis. In women the

cases have not infrequently been mistaken for *ovarian tumours*; but an ovarian tumour large enough to reach the ribs is central in position, while the history will show that it began in the lower part of the abdomen. On the other hand, the enlarged spleen may sometimes be felt in the pelvis behind the uterus. No absence of pallor should deter one from examining the blood, as the patient may have a good colour when the spleen is enormous and the leuchæmia pronounced; the presence of myelocytes, as well as the excess of polymorphonuclear cells and eosinophiles, are the distinguishing features of this disease.

The **Prognosis** is unfavourable, and recovery is uncommon.

Treatment.—Arsenic is the drug which seems to have given most promise; it must be used perseveringly and in increasing doses as long as it can be borne; and under its use the spleen has diminished in size considerably, and the leucocytes in number. Mosler has injected arsenic into the substance of the spleen. Inhalations of oxygen (one or two cubic feet daily) are believed to have done good. The local application to the spleen of the ice-bag, cold douche, or electricity (galvanic current) may reduce its size. During the last few years very striking results have been obtained by the application of Röntgen rays to the spleen, and to the epiphyses of long bones (femur), with the object of influencing the bone-marrow. The rays are applied every day or every other day for fifteen or twenty minutes, and the treatment must be continued for months. In several cases reduction of the leucocytes to the normal number and of the spleen to its normal size are reported, but relapses have occurred, and much has still to be learned as to final success.

Excision of the spleen is obviously unscientific, and has uniformly failed or been fatal from collapse or hæmorrhage.

LYMPHOCYTIC LEUCHÆMIA

(*Lymphatic Leuchæmia, Lymphocythæmia*)

This is of rarer occurrence than the myelocytic form, and the cases present much wider differences.

Condition of the Blood.—The feature of the blood is the immense predominance of the uninucleated non-granular leucocyte, or *lymphocyte*. These often form from 90 to 95 per cent.; sometimes the *small* lymphocytes are almost alone represented; in other cases they are all *large* lymphocytes. The *polymorphonuclear* cells form only from 2 to 5 per cent., and there are a very few *eosinophiles* and *myelocytes*. The red corpuscles may be about 3,000,000: nucleated red cells and mast-cells are rare.

Symptoms.—As contrasted with myelocytic leuchæmia, cases of this form are usually more rapid in their course, the spleen is rarely quite so large, and the various glandular organs all over the body, racemose as well as ductless, are often extensively involved.

It is also to be especially noted that, although the blood contains an extraordinary number of lymphocytes, the lymphatic glands, whether superficial or deep, are often not appreciably or much enlarged. The following groups of cases may be recognised:

First, those described as *acute leuchæmia*. This occurs in both sexes, and at all ages between seven and fifty-eight. Fraenkel mentions as antecedent conditions anæmia, pregnancy, injury, and some infectious diseases, especially influenza. The illness is fatal in from two to eight or nine weeks. It begins insidiously with general weakness and malaise, or pains in the spleen or joints. The external glands may enlarge, but are not always very prominent: there is slight enlargement of the spleen and liver, and the bones may be tender. A striking feature in many cases has been severe stomatitis with sloughing and gangrene of the gums; and with this there are fever, a high degree of anæmia, and hæmorrhages from the gums and bowels and under the skin. The lymphocytes are chiefly of the large variety.

Secondly, there are cases, almost equally deserving the title of acute leuchæmia, which are fatal in three or four months, and in which sloughing gingivitis or stomatitis is not a feature, and the lymphocytes may be of the small variety. In these cases also the external glands may not be very prominent, but many or all of the solid glands in the body are densely packed with lymphocytes, and hence considerably enlarged; for instance, the liver, spleen, kidneys, suprarenals, pancreas, salivary glands, and lachrymal glands, while the thymus persists and is greatly enlarged, and the cardiac muscle may be also infiltrated with lymphocytes. Exophthalmos has been also observed from leuchæmic infiltration of the orbital fat. More or less fever, hæmorrhages, and dropsy occur, and death soon follows.

There are, again, other cases in which the glandular enlargement occurs first of all, and the duration is from six months to three or four years. The lymphocytes are generally of the small variety, but this is not always the case. The lymphatic glands all over the body are affected, and they may be felt in the neck, groins, or axillæ. They are moderately large, not very hard, and move freely upon one another. The mesenteric glands are even more often enlarged than the above; the retro-peritoneal, thoracic, portal, and iliac glands less so. On section the glands are whitish-pink in colour, and microscopically are found to be distended with the lymphocytes. The bone-marrow is full of the same leucocytes, and the thymus and other organs may also be involved as above. In these cases, also, the spleen and liver may be very greatly enlarged. Sometimes the excess of lymphocytes in the blood is not apparent until a late stage, so that the glandular enlargement, with no excess of leucocytes, may simulate Hodgkin's disease (*see* p. 864).

The **Diagnosis** depends on a careful examination of the blood and enumeration of the leucocytes. It is in the lymphocytic form of leuchæmia, especially, that no changes in the blood-forming organs may be clinically recognisable: and in obscure conditions of

ill-health this fact should not be forgotten. The **Prognosis** is bad in the acute cases, in which there is little time for the operation of either arsenic or Röntgen rays. In less rapid cases these remedies should be tried.

ANÆMIA INFANTUM PSEUDO-LEUCHÆMICA

This condition was described by von Jaksch, and is found in infants and young children, under four years, but especially between seven and twelve months. There is anæmia with enlarged spleen, sometimes enlarged glands, and leucocytosis. The red blood cells are usually less than 60 per cent. of the normal, and poikilocytes and nucleated red cells (normoblasts) are present. The leucocytes number from 30,000 to 40,000; there is a slight relative increase of lymphocytes; and a few myelocytes (1 to 6 per cent.) are present. The hæmoglobin is much diminished.

The spleen is the subject of hyperplasia with moderate fibrosis. It is still a debated question whether these cases are different from, or only an extreme form of, the cases of moderate splenic enlargement with slight anæmia which are so commonly seen in infants, and which are often associated with syphilis and rickets. The condition of the spleen is not distinctive, and a small proportion of myelocytes in the blood is normal in young infants. In any case the condition is probably due to a toxin; if not that of syphilis, then it may be one produced in the intestinal canal in connection with digestive disturbances.

The **Treatment** consists in the correction of any dietetic irregularities and the use of mercury in cases suspected of a syphilitic origin.

CHLOROMA

Presenting close relations with lymphatic leuchæmia is the condition which has been called chloroma. In this there are numerous tumours, or lymphoid deposits, especially in the orbits (so that exophthalmos may take place), in the temporal fossæ and in the periosteum of the bones of the skull. Tumours may also grow on the conjunctiva and under the skin, and sometimes even during life these tumours have a green colour (*green cancer*).

The patients suffer from a cachexia similar to that of leuchæmia; there are prostration, anæmia, hæmorrhages into the retina and elsewhere, and optic neuritis. The blood presents a condition of lymphocythæmia; the lymphocytes reach from 70 to 90 per cent., they are often of the large variety, and a few myelocytes may be present. In a few cases myelocytes have predominated.

After death the various tumours are seen to have a green colour, which fades away on exposure; and the lymphatic glands, spleen, bone-marrow, and other organs are in a condition similar to that accompanying lymphocytic leuchæmia. The exact nature of the green colour is not known; it is not bile-pigment.

LEUCOCYTOSIS

Leucocytosis is the name given to a temporary increase of the white corpuscles either as a physiological event or in response to irritation by various infective toxins. The leucocytes rise to 15,000, 20,000, or 30,000, but rarely higher than this. The proportion of the leucocytes to one another remains as in health, or there is a slight excess of polymorphonuclear corpuscles. It occurs in pregnancy, and as a temporary event during the process of digestion (*digestion-leucocytosis*). The following are some of the diseases in which it is constantly found: Acute inflammatory diseases, suppuration from any cause, pyæmia, erysipelas, cerebro-spinal fever, pneumonia, pleurisy, empyema, phthisis, scarlet fever and rheumatic fever.

There are, however, diseases in which other cells than the polymorphonuclear are predominant; for instance, in typhoid fever, the small lymphocytes increase beyond 30 per cent. (*lymphocytosis*), and in malaria the large uninuclears reach from 11 to 20 per cent. An increase of the eosinophile cells (*eosinophilia*) occurs in some skin diseases, in asthma, and in connection with some parasitic diseases (filaria, bilharzia, trichina, ankylostoma, ascaris, hydatid).

LEUCOPENIA

Diminution of the leucocytes cannot be considered to be a disease in itself, but it has important relations with other changes in the blood. It occurs in long-continued fevers, and in different forms of anæmia; and is a constant feature of splenic anæmia, when the leucocytes may number only 4000 or 3000.

POLYCYTHÆMIA

It has already been stated (*see* p. 612) that in the cyanosis of congenital heart disease there is a great excess of the red corpuscles in the blood. This condition has been called polycythæmia, and has been found to occur in several other circumstances. In some of these the disease appears to arise from a primary change in the bone-marrow; they have been called *erythræmia*. In others, the increase appears to result from some disturbance in the circulatory or respiratory systems, requiring for compensation a larger number of carriers of hæmoglobin, such as congenital heart disease. These cases, from analogy with the reactive process of leucocytosis in infectious diseases, have been called *erythrocytosis*.

ERYTHRÆMIA

This occurs mostly in patients of between thirty and sixty years of age, though occasionally they are above or below these limits. In its pronounced form the red corpuscles vary in number from nine and

ten, to thirteen and even fourteen millions per cubic millimetre; and on standing, the corpuscles may be found to occupy nine-tenths of the volume of the fluid. The hæmoglobin is raised to 130, 160, or 180 per cent. of the normal.

The leucocytes are not always increased in number, but they may reach 24,000 per cubic mm. The total volume of the blood is estimated by Haldane's method to be much increased (*polyhæmia, plethora vera*), and it may be double the normal. The viscosity is increased to three or four times the normal. The specific gravity and the coagulation-time are not constantly either below or in excess of the normal. The blood-pressure is sometimes, but not always, high.

The conditions usually associated with this polycythæmia are *cyanosis,* and a moderate or considerable enlargement of the spleen.

The cyanosis is variable in different individuals, and in the same person may be much diminished, or may change to redness in a warm temperature. The mucous membranes are also cyanosed and the tongue may be blue.

The patients suffer from headache, lassitude, vertigo, dyspepsia, constipation, thirst, and various forms of hæmorrhage, which include epistaxis, bleeding from the gums, and menorrhagia.

Sometimes the arteries are sclerotic, and the urine may contain a little albumin. In some cases extensive venous thrombosis has occurred. In fatal cases the enlarged spleen has been found to be engorged, with some hyperplasia of the splenic pulp, but generally without evidence of erythroblastic or myeloid activity. The lymph-glands are generally unaffected: the liver may be engorged. The bone-marrow is generally deep red in colour, and no fatty marrow is seen, so that there appears to be a great increase in the function of red-corpuscle formation.

In accordance with this, the generally accepted view of the pathology of the condition is that from some cause or other the bone-marrow is stimulated to an excessive formation of erythrocytes; and that the other changes are secondary. There is little evidence in favour of other theories, namely, that the red cells are more durable than normal: or that the hæmoglobin has a diminished capacity for oxygen, and hence more corpuscles are required.

Differing somewhat from the above cases, which were first described as *polycythæmia with splenomegaly,* are some cases less frequently occurring in which the spleen is not enlarged, but the blood-pressure is greatly increased, and may even reach 300 mm. Hg. The patients are often turgid in the face, and may have hypertrophy of the heart, albumin in the urine, and signs of arterio-sclerosis. They were first described by Geisböck, who called them *polycythæmia hypertonica.* The patients are liable to apoplectic seizures, and may have chronic nephritis.

The progress of cases of erythræmia is variable; death has occurred from increasing cyanosis, or from cerebral vascular complications, or from tuberculosis

Treatment.—Venesection has temporarily relieved some cases. Splenectomy has been fatal and seems inadvisable; the effects of the Röntgen rays are doubtful. The general condition suggests a negative treatment on the lines of chronic Bright's disease: the avoidance of alcohol, high feeding, chalybeate drugs, and of such drugs as antipyrin, acetanilide, or phenacetin.

ERYTHROCYTOSIS

Under this term it is proposed to include the cases in which the polycythæmia is again due to increased activity of the bone-marrow (Parkes Weber), but this activity is stimulated by recognisable antecedent conditions. Thus one class is constituted by chronic cardiac and pulmonary lesions, of which congenital malformation of the heart is the most striking; and others are the various forms of acquired valvular disease, emphysema and chronic pulmonary diseases which may be accompanied by cyanosis. In these the deficient oxygenation of the blood is the stimulus to the bone-marrow. Another group is formed by the polycythæmia of persons resident at high altitudes, where the increase of erythrocytes compensates for deficient oxygen tension in the air available for respiration.

In both classes of cases the red corpuscles may reach seven, eight, or nine millions; but the numbers may be different in blood taken from arterial, venous, and capillary vessels. As in erythræmia, the hæmoglobin, the viscosity, and the total volume of the blood are all raised in amount; though the last statement, as regards the polycythæmia of high altitudes, is not so conclusively proved for man as it is for animals.

Polycythæmia also occurs in poisoning by phosphorus and carbon monoxide; it may be induced by injection of serum from an animal in which an erythroblastic reaction is in progress, and has been found in some infective diseases (syphilis, tubercle).

METHÆMOGLOBINURIA AND SULPHÆMOGLOBINURIA

Closely allied to the above are the cases of cyanotic decolouration caused by poisoning with the coal-tar products, antipyrin, acetanilide, and phenacetin. The appearance is chiefly due to the conversion of the hæmoglobin into methæmoglobin, and the red corpuscles are usually diminished; but in some cases it appears that polycythæmia occurs. A similar appearance is also due to the production of sulphæmoglobin. In some such cases the blood has been found to contain nitrites in considerable amount, and their presence has been attributed to the action of the colon bacillus in association with enteritis. The treatment suggested is the promotion of intestinal antisepsis; if necessary, by irrigation of the colon through a surgical opening (Gibson).

PURPURA

This term is applied to a diseased condition in which a number of hæmorrhages occur under the skin, so as to produce blotches of a more or less purple colour. It has been already seen that there are similar hæmorrhages in a number of diseases, either from an altered state of the blood, or from mechanical interference with its circulation; for instance, in scarlatina, measles, variola, typhus, cerebro-spinal fever, and the plague; in scurvy, hæmophilia, leuchæmia, Hodgkin's disease, cirrhosis, and acute yellow atrophy of the liver, and Bright's disease; in malignant endocarditis, and other diseases of the heart; and in some nervous diseases, *e.g.*, tabes dorsalis. As a direct result of poisoning from without, it arises from overdosing with potassium iodide (*iodic purpura*), and from the commercial use (handling and inhalation) of benzol or its chief constituent, benzene. In all these cases it is clearly recognised that a cause for the hæmorrhage exists; and this cause is often an infective toxin, or other poison. But in other cases the disease occurs spontaneously and without obvious antecedent in persons otherwise healthy.

The most familiar forms are *purpura simplex* and *purpura hæmorrhagica*, which appear to be essentially the same thing in different degrees of severity.

Symptoms.—In its mildest forms (*P. simplex*) purpura consists simply in the appearance of spots of a dull red, deep red, or bluish-purple colour in different parts of the body. They are circular, vary in diameter from a millimetre to a third of an inch, do not disappear on pressure, and are generally, when of this small size, not raised above the surface. They have no special relation to the position of the hairs. In some cases they occur only on the feet and legs, but in others are scattered uniformly, or, at any rate, indiscriminately. Each spot fades after a time, becoming brownish or yellow in tint, and the larger patches go obviously through the changes characteristic of a bruise. Very little constitutional disturbance accompanies the eruption; the patient may be pale, and loses appetite. Recovery generally takes place in from ten to twenty days.

In severe cases the hæmorrhages are more extensive, the skin may be raised by large masses of blood beneath it, and bleeding takes place from the various mucous membranes (*P. hæmorrhagica, Morbus maculosus Werlhofii*). The nose, mouth, stomach, and intestines, the kidneys, the female genital organs, and occasionally the bronchial mucous membrane may thus be the source of the blood. The gums are never swollen as in scurvy, but sometimes a spot of hæmorrhage is seen in their substance. If the loss of blood is considerable the patient becomes anæmic, and in the severer cases there may be some rise of temperature, and a stage of prostration ensues which terminates in death. Indeed, the

hæmorrhagic forms are very often fatal, and *post-mortem* examination may reveal other ecchymoses in nearly all the mucous membranes, in the pelvis of the kidney, in the pleura, pericardium, peritoneum, in the meninges, and even in the lungs and the medulla of the bones. A cerebral hæmorrhage may be the cause of death. Sloughing and ulceration of the mucous membrane of the bowels have also been found, leading to perforation and peritonitis.

Purpura rheumatica (*Peliosis rheumatica* or *Schönlein's disease*) is regarded by some as a hæmorrhagic erythema occurring during acute rheumatism, or in a rheumatic patient; while by others the arthritis is held to be the result of the purpura or its antecedent. Osler includes under the term any case in which purpura, erythema exudativum, purpura urticans, or urticaria, is associated with multiple arthritis. The illness may set in with sore throat, and pyrexia, occasionally causes endocarditis or pericarditis, and is very liable to relapse. Others limit the term to actual hæmorrhages, which may occur during rheumatism or in rheumatic subjects; beginning in the lower extremities, worse in the evening or reappearing then after improvement, persisting for long periods, but rarely fatal.

In *Henoch's purpura* the lesion of the skin, which may be erythematous or urticarious as well as hæmorrhagic, is accompanied by joint pains or swellings, attacks of abdominal pain, vomiting and hæmorrhage from the bowel, hæmaturia, and nephritis. It occurs in children, and recurs frequently during weeks or months. The sequence of symptoms in these cases varies a good deal, and the purpuric eruption is often late in its appearance, and not always very extensive. On the other hand, the early occurrence of the joint pains may give rise to a diagnosis of rheumatism, and in many instances the abdominal symptoms are the most prominent. Thus, abdominal pain, vomiting, and distension sometimes suggest intestinal obstruction or appendicitis: or the same symptoms with hæmorrhage from the bowel and a palpable tumour occurring in a child lead to a diagnosis of intussusception. Laparotomy has been performed in such circumstances: and sometimes an intussusception has been found, and easily reduced; or the supposed intussusception has proved to be a portion of bowel infiltrated with effused blood. The urine may contain much albumin, without blood or casts; or pure, or altered blood. Many cases are fatal; others recover, but in them the albuminuria may persist for months.

Pathology.—Little is really known of the pathology of purpura in any form. There is a decided diminution of the blood-platelets; otherwise the blood presents chiefly the characters usually found in secondary anæmia. Suggestive changes have been found in the vessels in some cases, and it appears that in some diseases associated with hæmorrhages the endothelium of the cutaneous capillaries is destroyed.

In some cases of benzol-poisoning there was marked oligocythæmia, leucopenia, and an aplastic condition of the bone-marrow (*see* p. 835).

Diagnosis.—In making the diagnosis, all the possible causes of a petechial eruption above mentioned must be excluded. *Scurvy* is distinguished by the spongy condition of the gums, the subcutaneous or fascial indurations, the greater degree of ill-health, and generally by its causation. *Malignant sarcomatous* growths may present some resemblance to purpura hæmorrhagica. It is well also to remember that the children of the poor sometimes present extensive petechial eruptions as the result of *flea-bites*. The spots are uniformly about the size of a pin's head, and all disappear entirely after a few days in better circumstances.

Treatment.—In milder cases, rest in bed, tonic medicines, and good simple food will often rapidly effect a cure. Iron, arsenic, and quinine may be given in the usual doses. Where the purpura affects the lower extremities chiefly it often disappears directly the patient takes to bed, and returns if walking about is too hastily resumed. In severe cases, also, arsenic may be given; but if hæmorrhage take place from the mucous membranes, astringents must be employed, such as turpentine, acetate of lead, ergotin, or dilute sulphuric acid. Turpentine (10 minims three times a day) is especially recommended for *P. rheumatica*. Calcium chloride and calcium lactate (20 grains every three or four hours, subsequently reduced to 15 or 10 grains) appear to have been of use, and should be tried. Since there is at present no clue to the nature of Henoch's purpura, its treatment can only be symptomatic.

SCORBUTUS

(*Scurvy*)

Scorbutus is characterised by a profound change in the blood, resulting from hæmorrhages under the skin and in other parts of the body, a spongy condition of the gums, anæmia, and prostration.

Ætiology.—It has been abundantly proved that the essential cause of this disease is the deprivation of fresh vegetable food. It may occur in either sex and at any age, and is clearly not contagious in its origin; but it has arisen over and over again in circumstances entailing a restriction of the dietary in respect of vegetables. Thus, it has been in past times the scourge of sailors on long voyages, so that it is frequently spoken of as sea-scurvy, though such a term does not now distinguish it from any other form; and it has severely affected armies and other large collections of individuals, such as those in prisons, and sometimes even in hospitals. Cases occasionally happen amongst those who could get vegetables, if for any reason, such as poverty, or dyspeptic troubles, they have habitually abstained from eating them. The influence of this wrong diet in producing the disease is aggravated

by several other depressing circumstances, which not infrequently co-exist, such as damp, cold, fatigue, drink, want of sunlight, as well as prostration from wounds, ague, dysentery, or syphilis.

Symptoms.—The disease generally comes on insidiously. The patient loses colour, becomes weak, languid, drowsy, or apathetic, and complains of flying pains in the loins or limbs. After a time—it may be a week or more—petechiæ appear upon the skin of the lower extremities and other parts of the body, and as a rule each hæmorrhage is situated around the base of a hair. The spots are small, red or reddish-brown, and not raised above the surface. Some others appear which more or less resemble bruises produced by violence, and large wheals or *vibices* may also be present. These various hæmorrhages occur all over the body; and there may be large extravasations of blood in the eyelids, or even subconjunctival ecchymosis, though very often the face is spared. Associated with this purpuric condition must be mentioned the occurrence of tense, brawny, indurated swellings in different parts of the body, especially in the popliteal space, the bend of the elbow, under the angle of the jaw, and in front of the tibia: these are due to effusions of blood, or blood-stained fibrin, or simply pale yellow fibrinous material, under the fascia, or between the muscular bundles, or between the periosteum and the bone.

Another feature which is commonly regarded as constant is the condition of the gums. These become swollen, fleshy, or spongy, detached from the teeth, and projecting beyond them in loose, bluish-red masses, which are painful, and bleed on the slightest touch. The teeth become loosened, the patient is unable to chew, and the breath is fœtid. The swelling of the gums may be so great that they project from the lips, and ulceration often results. The rest of the mouth is not affected in the same way. The tongue is large and indented. Sometimes the gums are not spongy, but only pale; and in all cases the change seems to be determined by the presence of teeth, so that it is absent where there is a gap in the series, and in toothless infants and old people.

When all these changes have developed, the patient has a sallow, bloated look, is markedly breathless on exertion, though no physical signs may be detected in the lungs; is subject to fits of syncope; and is totally unfit for bodily or mental exertion. The temperature, however, is generally not raised; the pulse is variable; and the urine is usually free from albumin. Hæmorrhage from the mucous surfaces, especially epistaxis, is not uncommon; and the feet are often œdematous.

In more serious cases there is hæmorrhage from the stomach and intestines, or from the lungs; pneumonia, gangrene of the lung, pericarditis, or pleurisy, which may be hæmorrhagic; or enlargement of the spleen and albuminuria. The skin over blood-extravasations may slough from pressure or irritation, and leave fungoid and very offensive ulcers. Dysentery sometimes complicates scurvy, but is generally regarded as having an independent

origin. The impairment of vision known as *hemeralopia*, or night-blindness, frequently occurs; the patient can see clearly and well in the daytime, but in the dusk, or the darkness of night, becomes quite blind, and cannot see his way about. It originates in a disturbance of nutrition of the retina; the ophthalmoscope shows no change in the eye, and normal sight is restored as the scurvy is cured.

Death takes place from increasing exhaustion, with anæmia and emaciation, generally after many weeks. But it may occur more quickly from sudden syncope, from pneumonia or gangrene of the lung, from hæmorrhagic inflammation of the serous membranes, or from cerebral hæmorrhage. In cases that recover, the improvement under suitable treatment is at once manifest, and often very rapid; but it is stated that the deeper effusions may leave thickening and fibrous bands, as a result of which the limbs are partly contracted and the corresponding muscles are atrophied. Sometimes the joints are ankylosed.

Infantile Scurvy.—Occasionally scurvy is seen in children under two years old, but it presents some differences from the adult disease, and is frequently associated with rickets, another dietetic disease. Scurvy is liable to occur in infants who are fed on highly sterilised milk, or on malted and other patent foods, and who do not have enough of, or any, fresh milk. Such children do not lose flesh, but become pallid; and then the limbs, especially the lower limbs, are affected, so that they do not voluntarily move them, and cry whenever they are touched or moved, or even when the hand is brought near them. The child lies often with the thighs abducted and the knees flexed, and so may be thought to be paralysed.

The bones are tender, there may be some œdema of the feet, and there may be swellings as a result of periosteal effusion; and the crepitus of a separation of the bones at the epiphyseal lines may be felt. If any teeth are through, there may be sponginess of the gums as in adults; and other hæmorrhages, such as epistaxis or hæmaturia, may occur.

Anatomical Changes.—In fatal cases of scurvy the lesions are found which have been mostly manifest during life—*e.g.*, the blood extravasations in the skin, and the effusions, whether sanguineous or fibrinous, in the aponeurotic sheaths, and under the periosteum; and in infants separation of the epiphyses. Occasionally hæmorrhage has occurred on the surface or in the substance of the brain. Frequently the pleural cavities contain serum, and there may be engorgement of the lung with serum or blood; sometimes it is even gangrenous. Hæmorrhages may also take place into the cardiac muscle, into the pericardium, or into the mucous membrane of the stomach and intestine: these latter may cause abrasion or ulceration. The liver and spleen are often large, much congested, soft, and friable; and an acute nephritis is described as occurring in severe cases. The blood presents a fall of red corpuscles to 4,000,000 or 3,000,000, or lower when hæmorrhages are abundant, a low colour-index, and generally no leucocytosis.

Pathology.—The exact pathology of scurvy is still an open question. In view of its connection with conditions of diet, attempts have been made to ascertain the particular constituent in vegetables whose presence is essential to health. Garrod believed it was the potassium salts, others that it was the organic acids; possibly both are necessary. Ralfe suggested that the disease was primarily a diminished alkalinity of the blood; and Wright's observations support this view. Others, again, believe that tainted, decomposed, or stale food develops a poison which acts on the blood; but the fact that sterilised foods cause infantile scurvy points strongly to the absence of some essential organic element.

Diagnosis.—There can be little difficulty in recognising this disease when the circumstances are such as have been known to lead to it; but the diagnosis requires care in isolated cases. It is distinguished from *purpura* by the general illness accompanying it, by the spongy gums, and by the deep-seated effusions in the hams and elsewhere. On the other hand, amongst the poorer classes of the population one may overlook mild cases, where the symptoms mainly consist of vague pains, with anæmia and ill-health, and the patients are likely to disregard a slight change in the gums or a few spots on the skin. An inquiry into the diet, or the administration of lime-juice or vegetables, will soon determine the nature of the illness. In infants it may be confounded with infantile paralysis, syphilitic epiphysitis, or periostitis.

Treatment.—This is essentially dietetic, the important point being the use of abundance of fresh vegetable food. Thus the patient should have a liberal supply of mashed potatoes, cabbage, greens, or salad, and he should also have some fresh meat once a day. When the mouth is sore, and the patient is unable to chew, milk, beef-tea, mutton-broth, and eggs may be given, and in any case, lime-juice or lemon-juice, to the extent of 3 or 4 ounces daily, diluted with water and sweetened to taste. Patients who are very ill must be carefully nursed, and kept in the recumbent position, and they may require brandy if the circulation is feeble. Drugs may be employed for the local conditions, but have no influence over the general illness. The gums are benefited by the local application of nitrate of silver in stick, or by alum, Condy's fluid, decoction of oak bark, and potassium chlorate and chlorine-water gargles. Diarrhœa, if present, may be met by bismuth or opium. For pain in the limbs, chloroform liniment may be used, and deep effusions have been treated successfully by friction with soap and water and the internal use of potassium iodide.

For the treatment of infantile scurvy, the same principles must be followed. Fresh milk boiled, not humanised or sterilised, potato well steamed and beaten up with boiled milk, raw-meat juice, grape juice, orange juice, lemon juice, and grated banana, are the foods which should be employed.

Prevention.—For the prevention of scurvy in time of war, or on board ship, or at stations where vegetables are scarce, Parkes

recommended, besides the use as far as possible of fresh and dried vegetables, that one ounce of good lemon-juice should be taken daily by each individual; that vinegar (½ oz. to 1 oz. daily) should be issued with the rations and used in the cooking; and that citrates, tartrates, malates, and lactates of potassium should be issued in bulk and used as drinks, or added to the food (in soups and stews, or as salt). The Shipping Act, 1894, enforces the first of these recommendations, at least as soon as the ship has been ten days at sea; and the lime-juice or lemon-juice must contain before being shipped 15 per cent. of palatable and proper proof spirit. No doubt the shorter voyages of steamships, as compared with sailing-vessels, have contributed to the diminution of scurvy.

HÆMOGLOBINURIA

Hæmoglobinuria means the presence in the urine of the blood-pigment, hæmoglobin, as distinguished from *hæmaturia*, the presence of blood itself, or, at least, the blood-corpuscles. Hæmoglobinuria arises when blood-corpuscles are broken up in the blood-vessels, so that hæmoglobin escapes into the plasma, giving it a pink tinge (*hæmoglobinæmia*), and is excreted by the kidneys. Possibly the excretion by the kidney does not occur if the destruction is confined to the blood in the portal circulation (*see* p. 834). A partial destruction (*hæmolysis*) of the corpuscles takes place under several circumstances: (1) The action of certain poisons, such as chlorate of potassium in large doses, pyrogallic acid, arseniuretted hydrogen, and naphthol. (2) The transfusion into one mammal of the blood of another; each kind of corpuscle becomes destroyed, and the serum is stained with hæmoglobin. In other words, the blood of one animal is hæmolysed by the serum of an animal of another species. (3) Exposure of the skin to extremes of temperature, such as burns or frost-bite. (4) The action of some fevers, so that a moderate degree of hæmoglobinæmia may result from scarlet fever or typhoid fever. (5) In blackwater fever (*see* p. 98). (6) In an *epidemic hæmoglobinuria* of infants (Winckel's disease). (7) The paroxysmal form of hæmoglobinuria, in which the altered condition of the serum has been also demonstrated. Under all these circumstances the plasma has a reddish colour, and the blood-corpuscles have little tendency to form rouleaux. Very pale corpuscles (*shadow corpuscles*) are seen, and the hæmoglobin is deficient.

In hæmoglobinuria the urine is red, dark-red, or reddish brown; it is acid, and deposits a dirty-brown sediment of epithelium, pigmented *débris* of corpuscles, perhaps casts containing blood-pigment, darkly stained urates, and opaque red granules of hæmoglobin. No blood-corpuscles can be seen. When examined by the spectroscope, the urine gives the two bands in the green and yellow characteristic of oxyhæmoglobin, and frequently another band nearer the red end of the spectrum, which is due to methæmoglobin.

The latter, which is an acid hæmoglobin, only appears after the acid urine has had time to act upon the oxyhæmoglobin; and if its action is continued acid hæmatin is also produced (Copeman). The urine always contains a proteid, either serum-albumin, or globulin.

PAROXYSMAL HÆMOGLOBINURIA

In this comparatively rare complaint, hæmoglobinuria occurs in isolated attacks.

Ætiology.—It is seen in young adults and middle-aged people up to fifty years of age, and is much more common in males than in females. In a few cases there is a history of malarial poisoning; in very many a history of syphilis; and rheumatism is said to be an occasional antecedent. Hereditary tendency does not play a prominent part. The most common immediate cause of an attack or paroxysm is exposure to cold, as from going out insufficiently clothed on a winter's day, or driving or walking far in the cold, or bathing in cold water. Exertion is another exciting cause.

Symptoms.—The beginning of the attack is marked in different cases by languor and weariness, a disposition to yawn, chill or rigor, pains in the limbs, nausea, vomiting, diarrhœa, and abdominal pain. Sometimes the fingers become white and cold, or the fingers, tip of the nose, and edges of the ears may become cold, livid, and even slough, constituting a condition of symmetrical gangrene, like that in Raynaud's disease (*see* p. 659).

The temperature may rise at the commencement, but soon subsides; and the whole duration of these symptoms is only from two to twelve hours. A slight enlargement of the liver and of the spleen is sometimes also observed. Either immediately after the first symptom, or only after three or four hours, the blood-coloured urine is passed, having the characters already described. But even this condition is only of short duration: in a few hours more the urine may be perfectly clear, and free from albumin and hæmoglobin; and in the intervals between the attacks it is always perfectly normal. Fagge pointed out that in some subjects of hæmoglobinuria, slighter chills are followed by transitory albuminuria. The effect of a paroxysm upon the blood-corpuscles is striking: whereas before an attack the red corpuscles are perfectly normal, after an attack they are found to present all shapes and sizes, with microcytes and a few megalocytes, and great variability of hæmoglobin-content, from excess to entire absence (*shadow corpuscles* or *ghosts*). Globules of hæmoglobin may be seen in the serum. Towards the end of an attack an icteric tinge of the skin is observed: and after many attacks in quick succession the patient becomes anæmic, the red corpuscles fall in number to 3,000,000, or 2,000,000, and the hæmoglobin to a somewhat less extent proportionately. Recovery of the red corpuscles takes place with remarkable rapidity.

Paroxysmal hæmoglobinuria is not in itself dangerous; but the

presence of renal cells and casts in the urine indicates that a nephritis is set up by the passage of the hæmoglobin; and in experiments with toxic agents producing hæmoglobinuria, the kidneys are found to be of a dark chocolate colour, from masses of the pigment collecting in the straight and convoluted tubes and in the glomeruli. The disease may last several years, and if the attacks are frequent the patient becomes anæmic, and acquires a sallow or faintly icteric tinge.

Pathology.—In paroxysmal hæmoglobinuria hæmolysis takes place within the blood-vessels, and the serum is stained with hæmoglobin; this escapes into the urine probably as oxyhæmoglobin, and by contact with acid urine is converted into methæmoglobin and acid hæmatin. In slighter attacks, probably only a small number of corpuscles are disintegrated, the hæmatin is disposed of in the liver, while the globulin alone is excreted in the urine: the proteid found in the urine in such conditions being indeed globulin and not serum-albumin.

The occurrence of the hæmolysis as a result of cold and other excitations has to be explained: and the view is put forward that there is a potential toxic *hæmolysin* in the blood of sufferers from this complaint, and that this consists of two parts, amboceptor, and cytase or complement. It is suggested that the action of cold is to cause the union of amboceptor with the corpuscle, and that subsequent co-operation of the complement with the return of warmth brings about solution of the corpuscles. The complement exists in normal blood, and it has been shown that the patient's serum will hæmolyse normal corpuscles, hence proving that there need be no specific liability of the corpuscles to break down.

The toxic origin of the hæmolysin is suggested by the fact that in the majority of patients there is a history or evidence of syphilis.

Treatment.—Exposure to cold must be carefully and systematically avoided, by the use of warm clothing, residence in warm rooms, and protection from night air as far as possible. It is much more difficult to diminish the susceptibility to its influence, which practically constitutes the disease. If there is any reason to suppose that syphilis is a predisposing condition, mercurials or iodides should be employed; if malaria is known to be an antecedent, quinine and arsenic should be tried. Their use must, of course, be continued for some time, and they may be given in daily doses of 5 or 6 grains of quinine, and 10 or 12 minims of the liquor arsenicalis. During the attack the patient should be made thoroughly warm by going to bed; and probably any renal irritation will be lessened by taking large quantities of fluid.

HÆMOPHILIA

Hæmophilia, or the *hæmorrhagic diathesis*, is a disease characterised by a tendency to excessive or uncontrollable bleeding, either spontaneous or traumatic. It is congenital, and very often hereditary, so that the subjects of the disease, often known as "bleeders," are the children of bleeders, and their brothers or sisters suffer perhaps from the same malady. Though it is most frequent and most severe in males, it is transmitted through the female, who may herself be entirely free from it; in this it resembles pseudo-hypertrophic muscular paralysis. Beyond this hereditary transmission no other cause is known.

Symptoms.—These generally appear within the first year of life, though they are sometimes delayed till the seventh or eighth year. In the most severe degree, spontaneous hæmorrhages occur from the nose, the gums, and the mouth, and less commonly from the stomach, the lungs, or the genitalia; they are sometimes preceded by a feeling of fulness. Bleeding from the nose is the most common and also the most fatal. Alarming and even fatal hæmorrhages may also occur after the most trivial operation, such as lancing the gums, vaccination, the extraction of a tooth, incision of an abscess, and the application of leeches, or after accidental wounds or a cut finger. In all these cases there is the greatest possible difficulty in stopping the flow of blood. Besides these losses, hæmorrhage takes place readily under the skin from slight blows, or even spontaneously, producing bruises or blood-tumours.

In the intervals between the bleedings the subjects of hæmophilia may appear to be in perfectly good health, but the enormous quantity of blood which is sometimes lost may cause a high degree of anæmia, which lasts for many months. Hæmorrhage also takes place into the synovial cavity of the joints, especially the knee-joint: this occurs most commonly between the ages of seven and fourteen, and results from blows, or from exposure to cold or to damp. The swelling and pain closely resemble those of rheumatism or synovitis, for which indeed the symptom has been mistaken. This joint affection is accompanied with fever; it may recover, but returns again and again. Eventually the joint may become ankylosed. A rheumatic affection of the muscles, and the occurrence of trigeminal neuralgia, are described as occasional complications of hæmophilia.

Three degrees of the disease may be recognised: one, common in men, and scarcely ever seen in women, in which there is a tendency to every kind of hæmorrhage, spontaneous or traumatic, interstitial or superficial; a second, in which spontaneous hæmorrhages from the mucous membranes only are present; and a third, which shows itself only by spontaneous ecchymoses, and which occurs amongst the women of bleeder families.

The majority of the subjects of hæmophilia die from loss of

blood before they are eight years of age; and though the chances of survival are greater after this period, even in middle age death may occur in the same way.

Pathology.—Apparently the extraordinary conditions of hæmophilia must either depend on the state of the vessels or of the blood. The viscosity of the blood is increased, its coagulation-time is prolonged, and there is leucopenia, but there is no other demonstrable change in its saline constituents, in its albuminoids, in its fibrinogen, or in its structural elements. Nor has the microscope revealed much alteration in the vessels; it has been noticed sometimes that the arteries are unusually thin. It should be stated that the bleeding takes place from the capillaries as a general oozing, and the blood is more often venous than arterial in colour. The fatty degeneration of the heart and of the arteries found in some cases is probably the result of anæmia.

Diagnosis.—It must be remembered that women sometimes suffer after puberty from a hæmorrhagic tendency, shown by ready bruising, menorrhagia, &c., who have never been bleeders in early life, are not descended from bleeder families, and do not transmit the tendency to their offspring. In the absence of exact information as to the nature of true congenital hæmophilia, the relation to it of these cases must remain doubtful.

Treatment.—Patients who are the subjects of hæmophilia should live on a light, unstimulating diet, and should pay particular attention to the bowels, any tendency to constipation being met by occasional laxatives. It is most important always to bear in mind the liability to bleed from any breach of surface; and the extraction of teeth, and all operations, large or small, should be avoided, unless absolutely necessary. The influence of drugs in ameliorating the condition is doubtful, but the tincture of the perchloride of iron is recommended, and calcium chloride has been given preparatory to tooth extraction with some success. If bleeding takes place, styptics must be employed in suitable doses, such as the perchloride of iron, ergot or ergotin, and gallic acid; more recently calcium chloride (15 to 20 grains) and the subcutaneous injection of gelatin (*see* p. 654) have been employed. The perchloride of iron may also be applied locally in solution, and in the case of bleeding after the extraction of a tooth, the crystals of this salt may be used to plug the cavity. Welch treated successfully twelve cases of hæmophilia in newborn children by the injection of normal human blood-serum. He recommends a dose of 10 c.cm. three times a day, or in severe cases a larger quantity every two hours. Considering the serious nature of this disease, and the manner in which transmission takes place through the female sex, it is clear that women who belong to bleeder families, even though themselves not the subjects of hæmophilia, should not marry.

DISEASES OF THE SPLEEN

The spleen lies in the upper part of the abdomen on the left side, and is entirely concealed by the ribs. In health its position and size can only be estimated by percussion. There is dulness in the left infra-axillary region over the ninth, tenth, and eleventh ribs, and the included spaces. In front this dulness is limited by a line drawn from the left nipple to the tip of the eleventh rib; behind it reaches nearly to a line continuous with the anterior margin of the latissimus dorsi. If the spleen becomes enlarged, it extends downwards and forwards, the dulness passes in front of the line above mentioned, and if the fingers be placed under the ninth and tenth costal cartilages while the patient takes a deep breath, the margin of the spleen can be felt to impinge against them. With greater enlargement, it comes distinctly below the ribs at this point, so that it can be readily felt, and occupies more or less of the left upper quarter of the abdomen. In extreme cases of leuchæmia the spleen reaches down to Poupart's ligament and crosses the middle line below the umbilicus, though it may remain on the left side above. An enlarged spleen is always dull to percussion, is continuous with the lower ribs, and is never overlaid by bowel or stomach. It descends along, and clings to, the abdominal walls throughout. The anterior margin is irregular, and presents one or two distinct notches.

The majority of the disorders of the spleen are secondary to other lesions elsewhere, and frequent reference has been made to its implication in different forms of fever and in disease of the heart. With some exceptions (leuchæmia, splenic anæmia, and hydatid) the lesion of the spleen is not generally a source of much trouble to the patient. The chief symptom which results from disorder of the spleen is *pain*, which may be present from the formation of infarcts, and from the resulting perisplenitis or abscess, but is not a marked feature in the enlargements which accompany fevers. There may be also a sense of weight from the great size the organ attains in some cases, especially in leuchæmia.

The pathological changes to which the spleen is liable, and the general symptoms which accompany them, will now be described.

Active Congestion.—The spleen is enlarged in many acute infectious processes, and this is most prominently the case in enteric fever, in relapsing fever, in ague and other malarial fevers, in pneumonia, pyæmia, malignant endocarditis, phthisis, and acute tuberculosis, and less so in puerperal fever, erysipelas, and syphilis. The capillaries and veins are distended with blood. The splenic pulp is swollen, and the capsule of the organ is distended. After death the spleen is found to be of dark-red or purple colour, and very soft; and the pulp is readily washed away by a current of water.

The histological changes resulting from infective processes in the spleen are, according to Muir, as follows: Great phagocytic activity of the cells of the pulp, especially non-granular hyaline cells and endothelial cells, which may be seen to contain numerous red cells, and neutrophile leucocytes: the presence of myelocytes in the pulp: apparent enlargement of the Malpighian corpuscles due to proliferation of cells around them.

Splenitis and Perisplenitis.—In some of these infective conditions the process goes beyond the stage of hyperæmia into one of acute inflammation, as shown, according to Ziegler, by the excessive quantity of white cells found within the vessels and pulp. Abscess is a very rare result of general splenitis. It more often results from the breaking down of infarcts in malignant endocarditis (*see* below). Accompanying the splenitis there may be inflammation of the capsule, *capsulitis*, or *perisplenitis*, with resulting adhesions to adjacent organs, or to the abdominal parietes. Acute or chronic capsulitis is very frequently found at *post-mortem* examinations; and its occurrence can often be traced, especially in the acute form, to infective processes.

Chronic Enlargement.—Splenitis may subside entirely, or go on to hyperplastic changes in the pulp, trabeculæ, vessels or capsule. The *ague-cake* already mentioned (*see* p. 99) is an example of this. A moderate enlargement is also seen in rickets, tuberculosis, congenital syphilis, and Hodgkin's disease; a much greater enlargement is often seen in ordinary cirrhosis of the liver, in malignant endocarditis, and in erythræmia; but the greatest size is reached in leuchæmia, in splenic anæmia, in infantile pseudo-leuchæmia, and in splenomegalic cirrhosis, in all of which the organ may seem to occupy half the abdomen.

The histological condition of the enlarged spleen has been often referred to under the above heads, and in different cases it results from thickening of the fibrous trabeculæ and septa, hyperplasia of the pulp, increase of the endothelial cells, great accumulation of leucocytes, of one or other variety, or venous engorgement from impeded return of the blood, as in cirrhosis and mitral disease. In some of these conditions it is difficult to say how far the enlargement of the spleen is a primary change, and how far it is the result of disease in other organs, such as the liver or bone-marrow. In splenic anæmia there is much evidence in favour of the spleen being primarily at fault: and this is supported by the favourable results of splenectomy in many cases. There are cases of chronic enlargement of the spleen with little or no anæmia, in which clinically no adequate morbid condition of the viscera or of the blood can be recognised. Possibly these are the early stages of some of the above disorders.

Passive Congestion.—The usual causes of venous congestion of the spleen are mitral valvular disease and cirrhosis of the liver; but in old heart disease the organ is often small and very hard.

Embolic Infarcts.—These are the results of the impaction of fibrinous particles detached from the valves of the heart or from thrombi in its cavities. The infarcts form wedge-shaped or conical masses, which may reach a large size, and occupy one-half or two-thirds of the organ. They go through the changes of colour elsewhere described (*see* p. 656), and in septic cases, such as pyæmia and malignant endocarditis, they become purulent, from the presence of pyogenic bacteria. Infarcts also occur in the spleens of leuchæmia and splenic anæmia.

Lardaceous Degeneration.—This change is similar to that which is seen in the liver and kidneys. It affects the splenic vessels and the Malpighian follicles, which last appear as gray specks upon the surface (*sago-spleen*); in other cases the lardaceous material is deposited between the cells of the pulp, and the organ is more uniformly pale. The diseased parts are coloured brown-red by the addition of tincture of iodine. The lardaceous spleen is enlarged, hard and smooth; the liver and kidneys are often affected at the same time.

Tumours of the Spleen.—*Benign* tumours and *primary carcinoma* are exceedingly rare. *Serous* and *sanguineous* cysts have been described. *Secondary carcinoma* occurs now and then in scattered deposits, and *lymphomata* are found in Hodgkin's disease. *Tubercle* appears in the spleen as a part of general tuberculosis, in the form of gray or often bright yellow nodules, which may reach the size of small peas, scattered throughout the substance and on the surface. Cases of primary tubercle of the spleen have been recorded. *Syphilitic* gummata are rare; but the spleen is often enlarged in congenital syphilis.

Parasites.—Exceptionally a hydatid cyst develops in the spleen and forms a large tumour.

Diagnosis.—From the frequency with which the lesion of the spleen is secondary to, or closely associated with, a disorder of some other organ or of the blood, it is obvious that the diagnosis of its disorders requires a careful investigation of the other organs, and especially a complete examination of the blood.

The **Treatment** of splenic lesions is dealt with under the various diseases with which they are associated.

DISEASES OF THE THYMUS GLAND

The thymus gland, of whose function little definite is known, though it probably has some relation to blood-formation, weighs from 7 to 10 grammes at birth, increases proportionately to the growth of the child until two years of age, and diminishes but little until the age of eight, ten, or twelve, but about the age of twenty has almost disappeared. Even in adult life, however, its remnant can be found if carefully sought, consisting of fibrous and fatty tissue, islets of lymphocytes, and a few Hassall's corpuscles (Dudgeon).

Its morbid changes are: atrophy in children, and hypertrophy or enlargement, or persistence with enlargement from various causes in adult life.

In atrophy, which is associated with marasmus, or tubercular and other chronic wasting diseases in children, the organ is reduced in size, and may weigh only 3, or 2 grammes, or less than 1 gramme. It is often white, soft, and œdematous; but the change is chiefly fibrous, there is an absence of the creamy pus-like, but really lymphocytic fluid found in the interior in health, the lymphoid tissue is scanty, and Hassall's corpuscles are degenerated and calcareous.

In acute disease in children, hæmorrhages, some fibrosis, and an increase of eosinophile corpuscles are often found.

Persistence, with enlargement, of the thymus occurs in a number of diseases, of which the most important are leuchæmia, especially the lymphatic form, Hodgkin's disease, exophthalmic goître and myxœdema, acromegaly, Addison's disease, myasthenia gravis, epilepsy, anæmia, rickets, scurvy, and the condition now known as lymphatism. In the last condition it may attain a large size, weighing often from half an ounce to one and a half ounces (15 to 45 grammes); but it has been known to weigh 6 ounces, and in a case of lymphatic leuchæmia described by myself more than a pound (586 grammes). The enlarged thymus in lymphatism has often a normal structure, except for the presence of numerous eosinophile corpuscles, of which the great majority are large uninuclear cells, while polymorphonuclear eosinophiles are in small number (Dudgeon).

The gland may be converted into a fibro-caseous mass as a result of tubercle, and it may be the subject of new growth.

THYMIC ASTHMA

This name signifies a dyspnœa, occurring in children, attributable to enlargement of the thymus. There is a good deal of difference of opinion as to whether an enlarged thymus necessarily presses upon the trachea; and this disorder appears undoubtedly to have been confounded with laryngismus stridulus (*see* p. 475), and with congenital infantile stridor (*see* p. 477). But independently of these, the balance of evidence seems to be in favour of such pressure sometimes occurring: though there may be a very large thymus without it.

If sudden death takes place without antecedent signs of dyspnœa, an explanation has probably to be sought in the disorder known as lymphatism (*see* p. 872).

DISEASES OF THE LYMPHATIC SYSTEM

The majority of the diseases to which the lymphatic system is liable arise as the result of the passage into the lymph-vessels of some substance foreign to them, such as micro-organisms, tumour-cells, or other solid particles, and the poisons of certain diseases which may ultimately prove to be of microbic nature. These either set up acute inflammation or cause a change in the gland of the same nature as the source from which the foreign substance has come. Thus we see the inguinal lymph-glands inflamed in syphilis; the glands of the jaw in diphtheria; the axillary glands in poisoned wounds of the arm; the bronchial glands in pneumonia; and the mesenteric glands in enteric fever. On the other hand, the bronchial glands become tuberculous as the result of phthisis; and the axillary glands are cancerous in consequence of carcinoma of the breast. Inflammation of the lymph-glands either goes on to suppuration, or subsides as the primary cause is removed, or becomes a chronic induration. Tubercle and cancer run the same course as they do in other parts. Hodgkin's disease, or lymphadenoma, and tuberculosis of the mesenteric glands will be separately described. The inflammation and suppuration of the cervical glands, which is often, but not always, a tubercular process, and probably depends on infection from the tonsils, are generally dealt with in surgical works.

The lymph-vessels are inflamed as a result of septic poisons, as, for instance, in *lymphangitis,* or absorbent inflammation, which involves the lymphatics between a wound and the nearest gland. Obstruction of lymph-vessels occurs under certain circumstances in tropical climates, especially by the nematode worms, *filariæ.* These conditions will be described under the name *Filariasis.*

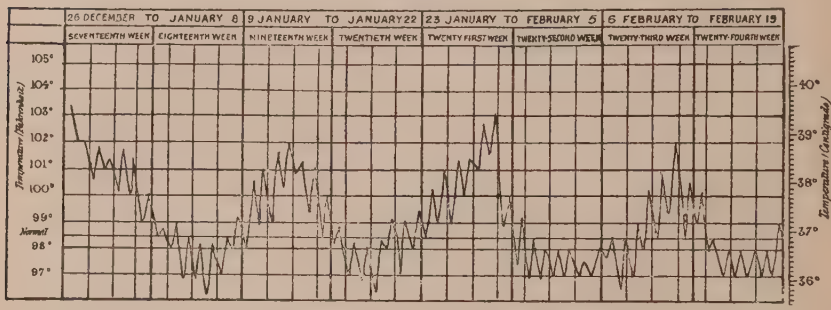
HODGKIN'S DISEASE

(*Lymphadenoma*)

Dr. Hodgkin first described, in 1832, a series of cases of enlargement of the lymphatic glands with a peculiar deposit in the spleen; and similar cases have been recorded by other writers as progressive multiple hypertrophy of the lymph-glands (Wunderlich), multiple malignant lymphoma (Billroth), and adénie (Trousseau). The name *lymphadenoma,* indicating the nature of the new growths which occur in the disease, is now mostly used in England, while German writers employ the term *pseudo-leuchæmia,* from its apparent relations to leuchæmia. It has also been called *anæmia lymphatica.*

Ætiology.—Very little is known of its causation. It occurs at all ages, but is most frequent in early and late adult life. Men are more often attacked than women. In a few cases depressing causes, such as intemperance, insufficient food, exposure to cold, and parturition have been noted; and in a good many instances the disease seems to have been brought about by a local lesion, such as a blow, a discharge from the ear, an abscess or an eczema,

FIG. 67



Chronic Relapsing Pyrexia in Hodgkin's Disease.

in consequence of which a group of glands is irritated and enlarges, and subsequently the other glands in the body are involved. More often, however, no cause whatever can be discovered.

Symptoms.—The chief clinical features of the disease are enlargement of the lymphatic glands and anæmia. Generally, the *lymphatic enlargement* occurs first, the change beginning in the cervical glands in most cases, and subsequently involving those of the axilla and the groin. The glands form irregular and nodulated masses of different sizes, extending, perhaps, from the clavicle to the jaw, or of such a size in the axilla as to prevent the arm being applied to the side. The individual glands are as large as a pigeon's egg or a hen's egg, are either soft or firm, usually painless, and at first freely movable upon one another under the skin. Subsequently they may become adherent, but rarely caseate or suppurate. Other smaller groups of glands are also involved, such as the occipital; and the change affects the glands in the interior of the body—namely, the bronchial, mediastinal, mesenteric, and retro-peritoneal. In many of these regions the growth of the glands may be such as to cause serious pressure on the neighbouring parts. These are in the neck, the larynx, trachea, and œsophagus; in the thorax, the large veins, and the recurrent nerves; in the abdomen, the nerves of the solar plexus; and in the axillæ and groins, the nerves supplying the limbs.

The *spleen* is, as a rule, only moderately enlarged; it projects a little below the left costal margin, or occupies the left upper quarter of the abdomen; it rarely attains the same size as that seen in myelocytic leuchæmia.

Anæmia is a prominent symptom, even comparatively early, in Hodgkin's disease. The red corpuscles are diminished to 60, 50, or 40 per cent. of the normal; the hæmoglobin is diminished to a still greater extent (chlorotic type); and in severe cases poikilocytes and nucleated red cells (normoblasts) are seen. The leucocytes are generally not increased in number, but when the glands are soft (or inflamed) they may be as many as 15,000 or 20,000 per cubic mm. Of these the polymorphonuclears form about 70 per cent.; lymphocytes are not numerous, but eosinophiles are sometimes in excess, both relatively and absolutely. If, as may happen, a pronounced lymphocytosis develops, the case would be regarded as a lymphocytic leuchæmia (*see* p. 842).

Pigmentation of the skin sometimes occurs, and is ascribed to pressure on the solar plexus by enlarged glands; occasionally there is intense *prurigo*, with the formation of papules. The urine is only occasionally albuminous.

Pyrexia.—The temperature is often raised, and is either continuously above normal, or shows daily remissions, or periods of fever from 7 to 10 days' duration, alternating with similar periods of apyrexia (*see* Fig. 67). Some cases published under the name of *Ebstein's disease*, or *recurrent pyrexia*, have been cases of Hodgkin's disease affecting the internal glands chiefly or only.

A certain amount of weakness is soon observed, and as the disease progresses the effects of the anæmia become more pronounced. There is generally a good deal of dyspnœa, partly from the anæmia, and partly from mechanical interference with the trachea, bronchial tubes, or lungs. In time, also, œdema of the lower extremities takes place, with, perhaps, ascites, pericardial effusion, or hydrothorax; and hæmorrhage from the nose or gums, or under the skin, may occur as in other severe blood-changes. Finally, death is caused by exhaustion, suffocation, or starvation from the pressure of enlarged glands, by hæmorrhage, by cerebral disturbance, coma or convulsions, by pneumonia, pleurisy, or œdema of the lungs. The duration is generally several months, or one or two years; occasionally the cases are much more rapid.

Anatomical Changes.—On *post-mortem* examination, it is found that there are two important changes: one, the enlargement of the ordinary lymph-glands; the other the growth of tumours having a similar (lymphadenomatous) structure, in different organs of the body, and in subserous and other tissues, probably in most cases arising from natural lymphatic formations. The consistence of the glands varies in different cases of lymphadenoma. They are light gray, or grayish-white on section, and the softer kinds yield, on scraping, a turbid juice, consisting mainly of lymphocytes. Microscopic examination shows that the enlarged gland consists of lymphocytes or lymphoid corpuscles in a nucleated reticulum; and in the harder varieties this reticulum is abundant, and the corpuscles relatively less, while in the softer varieties the corpuscles are abundant and the reticulum is very fine. In addition to the

numerous lymphocytes there are endothelial cells, often with karyokinetic figures, giant cells with one or more nuclei, and sometimes eosinophile leucocytes, which may also be uninucleated or multinucleated. Adhesion of the glands to one another is caused by the lymphoid corpuscles invading the capsule of the gland, and infiltrating the interglandular tissue. Occasionally, as already stated, a little caseous change may occur in a few glands.

The lymphadenoid growths which are independent of the glands proper occur in the spleen, kidneys, liver, lungs, and other parts. The spleen is generally larger than normal, and may weigh even 30 ounces; it is moderately firm, and presents on section a number of white or yellowish tumours, from one-eighth to half an inch in diameter, scattered through its substance, giving it an appearance which has been compared, not inaptly, to "hard-bake." These tumours arise from the Malpighian corpuscles, and consist of lymphoid corpuscles in a reticulum. Similar tumours occur scattered through the liver or the kidneys, which may also show a more diffuse growth of lymphoid tissues; and swelling due to similar growth may occur in the tonsils, and in the follicles of the pharynx, stomach, and intestine. Nodules of growth are also present in the lungs, and soft pinkish-gray, flat masses, having a similar structure, have been found under the pleura and other serous membranes. In one case under my care, growths the size of a pea had formed in pleural adhesions, which stretched across the fluid of a hydrothorax. The epididymis and testicle have been invaded. Sometimes in this disease, also, the bone-marrow is affected, being converted into a reddish-gray, semi-diffluent matter.

Pathology.—This is still very obscure; but the resemblance in the organs affected to the conditions of lymphocytic leuchæmia strongly suggests a pathological alliance. There appears to be little justification for the view that lymphadenoma is only a special result of tubercular infection.

Diagnosis.—It may be difficult to distinguish the early stage of Hodgkin's disease of the glands from tubercular enlargement, especially when the growth is confined to one set of glands. *Tubercular changes* affect more often a single group, occur in young people, and lead to caseation and suppuration. *Cancer* of the glands may resemble Hodgkin's disease, but secondary deposits are not so likely to take place in other glands as in the different viscera. A general glandular enlargement in *syphilis* can usually be distinguished by the history and by the influence of drugs. If a leucocytosis develops, its extent and the characters of the differential count will require careful consideration (*see* Leuchæmia).

Treatment.—Arsenic has been of great benefit in some cases: it should be given in increasing doses until as much as 15 minims of the liquor arsenicalis three times daily is being taken. The more usual tonics—iron, cod-liver oil, quinine, and others—have no influence. In cases where the enlargement has been confined for some time to one set of glands, their excision has sometimes

appeared to postpone the end, but in most cases the disease has spread in spite of it. Great improvement has been recorded in a few instances from exposure to the Röntgen rays, which appear to act prejudically upon lymphoid tissues : but relapses are almost certain. And, in fact, few cases of complete recovery are known to have occurred.

TUBERCULOSIS OF THE MESENTERIC GLANDS

(*Tabes mesenterica*)

Ætiology and Pathology.—The mesenteric glands undergo the same changes of tuberculisation, caseation, calcification, and occasionally suppuration, as are seen in the bronchial glands (*see* p. 664). Similarly the process may be secondary to a tubercular lesion of the intestine—namely, the tubercular ulcer, or it may be associated with tubercular peritonitis; but it is not infrequently, especially in young children, a primary lesion. The glands are increased in size, and form large masses, which may become perceptible on examination of the abdomen. As a secondary result they cause chronic and tubercular peritonitis; and in the event of suppuration, the abscess may burst into the peritoneum, and set up a general acute inflammation. The disease is much more frequent in children than in adults, and there is a great probability that it is caused sometimes by the ingestion of milk infected with tubercle.

Symptoms.—When we consider that the mesenteric lymph-glands are those in connection with the lacteals, it will be readily understood that any extensive disease in them will seriously interfere with nutrition. Patients with mesenteric disease are wasted; and the small arms, legs, and chest contrast strikingly with the abdomen, which is large, partly from the increase in size of the glands, and partly from the distension of the intestines by flatus. The enlarged abdomen is resonant, and the distended bowels mostly prevent the glands from being felt. Sometimes, however, they are of sufficient size to be recognised by the hand. The accompanying symptoms are diarrhœa with griping pain, and febrile action with malaise. The motions are brown, watery, and offensive. If tubercular ulceration is also present the diarrhœa is likely to be more constant, and the motions are sometimes lighter brown or yellow. Peritonitis is indicated by more pain, tenderness, and abdominal tension.

Diagnosis.—There are three conditions in children, characterised by wasted limbs and a large abdomen, which are liable to be spoken of as mesenteric disease and "consumption of the bowels." They are tabes mesenterica, tubercular peritonitis, and simple indigestion with diarrhœa. Of these the last is the most common. The diagnosis of tabes can rarely be decided upon unless the enlarged glands are felt, and this is usually prevented by the inflated

bowels. Their existence may be suspected if there is continued febrile action, and a tubercular family history; and if the symptoms persist in spite of such changes in the diet as would probably cure simple indigestion or diarrhœa. The tuberculin tests may, of course, be applied (*see* p. 535).

Prognosis.—This is not necessarily unfavourable, so long as the change in the glands is not complicated with ulceration of the bowel, or tubercle elsewhere, or extensive chronic peritonitis.

Treatment.—The implication of the lacteals has suggested that the food should not contain too much of a fatty nature. Thus, animal food should be given more abundantly, and what milk is used should be boiled or sterilised. The effect upon the diarrhœa must, of course, be carefully watched; and this may be met with astringents, such as aromatic chalk powder, catechu, Dover's powder, or dilute sulphuric acid. The same tonic remedies should be given from time to time as in bronchial phthisis; but cod-liver oil is not so useful here, on account both of its fatty nature and of the diarrhœa when present. Calcium chloride may be tried in doses of five grains, given in milk.

FILARIASIS

Nine or ten distinct species of the nematode worms *Filiariæ* occur as parasites in the human body, of which some infest the connective tissues, and others the blood-vessels and lymphatics. The filariæ occur extensively in tropical and sub-tropical countries, especially in West Africa, India, the West Indies, and the South Pacific Islands. The most important is that of which the embryonal form is known as *Filaria sanguinis hominis nocturna*, and the parent worm as *Filaria Bancrofti*.

Life-history of Filaria nocturna.—The adult female is filiform, from 3 to $3\frac{1}{2}$ inches long, and $\frac{1}{100}$ to $\frac{1}{90}$ inch in thickness; the male is much smaller. They have been seen in dilated lymphatic vessels, and there the female discharges an innumerable progeny of embryos, which are sufficiently minute to pass through the lymphatic channels into the thoracic duct, and thence into the blood-vessels, where they are found during the life of the host in extraordinary numbers. These embryos are also filiform, about $\frac{1}{90}$ inch long and $\frac{1}{3200}$ inch broad; they are contained in a soft covering, in which they elongate and shorten themselves, and which commonly projects for some little distance beyond the caudal extremity. This covering is conjectured to be the shell of the ovum which has been stretched by the growth of the embryo. The embryos are extremely active, twisting and wriggling amongst the blood-corpuscles as they are seen on the slide of the microscope.

In the case of the *F. nocturna*, the embryo filariæ can only be found in the blood-vessels of the skin during the night, from 6 or 7 P.M. to 8 or 9 A.M., whereas during the daytime they are in very small numbers or entirely absent. It is conjectured that they must

during the daytime occupy the blood of the deeper vessels; and it has been shown that this diurnal variation depends on the habit of the host, with regard to waking and sleeping, and not upon light and darkness. In the tropics, the presence of these embryos in the cutaneous vessels at night-time is of importance to their future development, as, like some other entozoa, they require an "intermediate host" between the stage of embryo and that of complete development. Such an intermediate host is the mosquito, which, while sucking the blood of its human victim, draws up the filariæ into its stomach. Within a few hours of their ingestion, the embryos escape from their sheaths, and pass from the stomach of the mosquito to the thoracic muscles; and here they both develop their organs and grow to a considerable size during the period of six or seven days which are required for the mosquito to deposit her ova on the surface of the water and die. Thereupon it seems that the filariæ, now about one-sixteenth of an inch in length, get into the water and are able to infect human beings by being swallowed when the water is drunk. Ultimately the filariæ establish themselves in the lymphatic vessels of their host, become sexually mature, and furnish the embryos which are found in the blood.

Pathological Relations.—The embryo filariæ may be found in the blood of perfectly healthy persons, and no symptoms appear to attach to their presence therein. They may be readily seen with a low power of the microscope. A film of the blood may be spread on a microscope slide, and examined at once or allowed to dry. Manson recommends staining with fuchsin (three or four drops of saturated alcoholic solution to an ounce of water) without previous fixing, and examination without cover-glass.

It is the presence of the adult parasites in the larger lymphatic vessels which gives rise to definite pathological conditions; and this is as a result either of blocking of the channels by masses of the parasite, or more often by inflammatory changes in the walls of the vessels (*lymphangitis*), which their presence excites.

Thus are produced obstruction of the thoracic duct or other lymph-vessels, and in consequence varicosity of more remote vessels, and the solid œdematous condition of which *elephantiasis* is probably an example. The obstruction to which the thoracic duct may be subject was well shown in a case recorded by S. Mackenzie. He found a large mass of dilated lymph-sinuses and glands, extending from the bifurcation of the aorta to the diaphragm, and occupying all the space between the two kidneys. The lower part of the thoracic duct was sinuous and pouched; and above the diaphragm it became impervious, and was lost in the tough, dense material, apparently of inflammatory origin. If the thoracic duct is obstructed, the chyle-vessels and lymph-vessels below it become dilated and varicose, and the chyle, unable to pass by its usual course, will go along new channels, and thus come into relation with such parts as the walls of the bladder, the tunica vaginalis, or the pleura. By the rupture of dilated lymph-vessels in connection with these

parts, chyle or a chylous fluid is extravasated into the hollow cavities, and thus is explained the occurrence of *chyluria*, chylous hydrocele or *chylocele*, *chylous pleurisy*, and *chylous ascites*.

The connection of elephantiasis with filariæ is not so certainly shown, for the blood in these patients is generally free from embryo filariæ; but elephantiasis occurs in precisely the same parts of the world as filarial disease. It is quite clear that elephantiasis is due to lymphatic obstruction, and Manson thinks that from time to time the parent worm, perhaps from injury, gives off prematurely the ova, which are shorter, thicker, and more rigid bodies than the ensheathed embryos hitherto described, because the coverings have not yet yielded to the active movements or growth of the contained embryo; and that such broad ova, which may in course of time be numerous, obstruct the lymphatic channels sufficiently to produce these results, and also by accumulating in the glands prevent the passage of embryo filariæ into the blood-current.

Some of the more important results of filarial obstruction of the lymphatic vessels will now be described.

CHYLURIA

The urine is opaque, whitish, or milky in appearance, and has an odour of milk. On standing, a layer of fat may collect on the surface; and generally, also, a soft coagulum forms, which is either transparent or opaque. If it is shaken up with ether, and placed under the microscope, the turbidity is seen to be due to minute oil globules and granules. It contains also a small quantity of albumin. Sometimes blood is present, and gives the urine a pink or darker red colour. Careful microscopical examination of the sediment will often detect the embryo filariæ, which have been already described as being present in the blood, and which have obviously escaped with the lymph or chyle into the urinary passages.

Chyluria, when once established, is not constant; it may disappear and again recur. When present, it is generally more marked after a meal.

There is not necessarily any disturbance of the health; the patient may seem perfectly well. On the other hand, there is often some uneasiness or pain in the back, loins, or perinæum; and it may be febrile symptoms, debility, or mental depression. Retention of urine, from blocking of the urethra by fibrinous coagula, is sometimes the first sign. In prolonged cases—and it may last twenty or thirty years—there are emaciation, craving appetite, and severe thirst. The disease occurs at all ages, and equally in males and in females.

Diagnosis.—Chyluria is readily recognised, and if it occur in persons from the tropics, the filariæ may be looked for in the blood and the urine. But chyluria and the other lymphatic lesions are occasionally seen in persons who have never resided abroad, and may be exceptionally due to obstruction or rupture of lymphatic

vessels produced in other ways. For instance, Whitla records a case of chylous ascites from invasion of the thoracic duct by tubercle.

Treatment.—When once established, little can be done beyond supporting the patient's strength as much as possible, so as to meet the drain of fat, fibrin, and albumin through the urine.

Manson relies on rest in bed, elevation of the pelvis, restriction of food and drink, and gentle purgation, for at least temporary relief.

LYMPH-SCROTUM

Numerous clear vesicles appear on the scrotum, which is enlarged, soft, and spongy; the vesicles may give way and discharge a fluid, which is obviously lymph. The inguinal glands are enlarged, and there is a liability to the erysipelatoid attacks or fever which occur in elephantiasis. Embryo filariæ may be found both in the blood and in the lymph from the vesicles.

ELEPHANTIASIS

The legs and the scrotum are the parts most commonly affected. It is not necessarily symmetrical; it may affect one leg alone (*Barbados leg*), or the leg below the knee only, or the scrotum, ears, lips, arm, or the lower part of the abdominal wall. If the leg is affected, it becomes enlarged to two or three times its natural size. The skin looks œdematous, but it does not pit on pressure; it is obviously greatly thickened. Where folds naturally occur, as about the knee or ankle, there are deep sulci, and these may be moist from retained sweat or sebum. Pigment is increased in the limbs, the surface becomes rough and scaly, here and there are patches of hypertrophied papillæ, and, in other parts, vesicular or moniliform prominences due to dilated and varicose lymphatic vessels. Sometimes these burst and discharge a more or less turbid lymph. The scrotum has sometimes grown to a tumour weighing more than one hundred pounds.

In tropical countries the disease often begins by attacks of lymphangitis with swelling and redness like erysipelas, accompanied by fever (*elephantoid fever*), after the subsidence of which the leg is left bigger than normal. A fresh attack of fever and local inflammation after some months again leaves the leg worse than it was after the first, and the evil goes on increasing. Sometimes, however, the enlargement is gradual, and not at any time associated with febrile attacks.

In England erysipelas and phlegmasia alba dolens are occasionally causes of a permanent elephantoid change. Local pressure on veins and lymphatics may cause it, and I have seen it as a part of extreme obesity.

Anatomy.—The change affects chiefly the subcutaneous layer, which is enormously thickened by the growth of new connective tissue, partly gelatinous, but mostly in dense fibrous bands. The blood-vessels and lymphatics and lymphatic glands are much

enlarged, and sometimes the nerves. The corium and epidermis are only slightly affected.

Treatment.—Very little can be done, except surgically; thus a large scrotum can be removed. But in any other part of the body such a measure is too serious. Bandaging, especially with rubber bandages, and elevation of the limb, may give temporary relief. Ligature of the artery has been tried and failed.

OTHER FORMS OF FILARIASIS

The embryonal forms of *filaria diurna* are found in the blood in the daytime, and disappear at night; those of *filaria perstans* are in the blood both day and night. The parental forms of these are not with certainty known. *Filaria medinensis* is well known as the *guinea worm,* which infests the subcutaneous tissues of the legs in certain parts of India and tropical Africa. This, the female worm, is from 12 to 30 inches in length, and only one-tenth of an inch in width. It discharges its embryos into water, and they find an intermediate host in the small crustacean *Cyclops quadricornis.* It appears probable that it is transferred to man by means of drinking-water.

LYMPHATISM

(*Status Lymphaticus*)

As is well known, sudden death not unfrequently takes place in the course of chloroform anæsthesia, when it is almost certain that no undue amount of the anæsthetic has been administered. When such cases occur in children, it is often found that there is a general hyperplasia of the lymphatic structure throughout the body, enlargement of the tonsils, Luschka's tonsil (adenoids) and the follicles at the back of the tongue, the lymph-glands in the neck, axillæ, and groin, and the solitary follicles of the intestine; and it may be the thyroid gland, spleen, and liver. More striking than these may be the persistence and enlargement of the thymus gland, which has been already described as an important event in lymphatism (*see* p. 862). Hyperplasia of the red bone-marrow, and hypoplasia of the heart, and narrowing of the aorta have been found in some cases. Rickets is often present, and sometimes the children have been previously out of health, or have had convulsions.

Sudden death, however, occurs in children, the subject of such pathological changes, independent of anæsthesia: and its occurrence in either case is difficult to explain. It is attributed by some to pressure of the thymus on the trachea, by others to toxæmia, to a defective condition of the blood, to intravascular clotting, to diminished powers of resistance, or to reflex nervous influence.

The pathology of lymphatism is very obscure: and the relative share of the thymus and the lymphatic structures difficult to estimate. The change in the thymus is the predominant feature

in most cases; and the disease has been ascribed to an excessive secretion from this gland exciting a general lymphatic hyperplasia.

Diagnosis.—It is, of course, very desirable to be able to recognise the condition before administering an anæsthetic. There may be dulness over the sternum and the costal cartilages on either side, but it is often absent; or the thymus may be recognised by the Röntgen rays. The size of the above-mentioned lymphatic structures, where they are accessible, should be ascertained.

The **Treatment** of status lymphaticus has been attempted with the Röntgen rays applied to the thymus, apparently with some success; and by operations upon the thymus, raising it by sutures, or removing it altogether.

DISEASES OF THE THYROID BODY

Besides the racemose secreting glands provided with ducts, and the glands or organs connected with blood-formation (spleen, lymph-glands, and possibly the thymus), there are certain bodies with special histological structure, the diseases of which are accompanied by very striking changes in the metabolism of the body. From the evidence derived from these diseases, as well as on experimental grounds, it is believed that these organs elaborate and pour into the blood a substance or substances (*internal secretion*) which have an important influence, either supplying something which is necessary to the economy, or neutralising or destroying other bodies which are harmful. These organs are the thyroid body (and parathyroid bodies), the suprarenal bodies, or adrenals, the pituitary body, and the carotid and coccygeal bodies. Further it is believed that some glands with an external secretion also provide an internal secretion, such as the pancreas, which has an important relation to diabetes, the kidneys, and the testes.

Atrophy of the thyroid and its removal by operation in men and animals are followed by a certain train of symptoms known as *myxœdema;* and a very similar condition, *cretinism,* occurs in young persons from congenital absence or disease of the thyroid. Atrophy and tubercular disease of the suprarenal bodies leads to the group of symptoms known as *Addison's disease;* and tumours of the pituitary body cause an extraordinary growth of bones, known as *acromegaly.* These are regarded as probably due to the suppression of the internal secretion in each instance. In the case of the thyroid gland a pathological condition is known, associated with symptoms (*Graves' disease*) which are the converse of those seen in myxœdema, and it is assumed that here the internal secretion is provided in excess.

Of late much interest has centred about the parathyroid glands,

which are small bodies, from four to six in number, situated near, or within, the substance of the thyroid, and consisting of groups of cells in a fibrous meshwork, but without the vesicular arrangement and colloid contents of the thyroid. Changes in these bodies—fatty degeneration, fibrosis, cyst and colloid formation—take place in the course of various diseases, especially the thyroid lesions presently to be described, Addison's disease, leuchæmia, pernicious anæmia, and diabetes. No constant symptoms or groups of symptoms can be shown to depend on these lesions (Forsyth).

BRONCHOCELE

(*Goître*)

A bronchocele or goître is an enlarged thyroid gland.

Ætiology.—The most prominent ætiological fact is that goître is frequent in certain localities, and especially in limestone districts. In England it occurs in Derbyshire and in Devonshire. On the Continent it is frequent in the mountainous regions of Savoy, Switzerland, Northern Italy, the Tyrol, and Styria. This has suggested some influence of the limestone formation upon the water which the inhabitants drink. But whether the agent is the dissolved minerals or some other substance is open to question. Captain McCarrison, from experience in the Chitral and Gilgit valleys, concluded that the cause could not lie in excess of solids, or of hardness, or calcium, magnesium, or iron compounds; but that it must be some suspended micro-organisms. Goître was produced in fifteen days in previously healthy individuals by the administration of the suspended matter separated by filtration from these waters; but no enlargement of the thyroid occurred when the separated matter was boiled before administration. Bad ventilation, overcrowding, and accumulated filth have always been associated with goître; but they are not peculiar to goîtrous districts. In England, goître is most frequent in young women.

Endemic goître both in Central Europe and in India is often found to be associated with *cretinism.*

Morbid Anatomy.—Goître occurs in the following forms: (1) A simple soft hypertrophy or parenchymatous goître; (2) encapsuled adenomata, which may be solid or cystic; (3) malignant disease; (4) exophthalmic goître (*see* p. 875). Simple parenchymatous enlargements are mostly bilateral; markedly unilateral swellings are either adenomatous or malignant. In both parenchymatous and encapsuled forms fibroid changes may take place; but parenchymatous swellings may be very hard to distinguish from distension of the follicles with colloid material. The fibrous tissue results from hypertrophy of connective tissue between the follicles; the cysts result from distension of the follicles of the gland. The size varies from a moderate prominence of the neck on either side, to a mass as large as the fist or a fœtal head, which

hangs down in front of the upper part of the sternum, such as those which have been so common in Switzerland and Savoy.

Symptoms.—Enlargement of the neck and a feeling of fulness are often the only symptoms. If the goître is very large, there may be dysphagia from pressure on the œsophagus, or dyspnœa from compression of the trachea, or of the recurrent laryngeal nerves. Malignant disease occurs generally after middle age, and forms a hard, rapidly growing tumour, which infiltrates and presses upon surrounding parts.

Diagnosis.—This is, as a rule, easy. The thyroid nature of any enlargement is proved by its movement up and down with the larynx during the act of swallowing.

Treatment.—The patient should remove from the locality where the disease is known to be prevalent, and every hygienic condition should be made perfect; the enlargement has entirely subsided in some cases when distilled water or rain water was drunk instead of that in common use. Iodine has a marked influence upon goître; and should be given internally, as tincture with potassium iodide; or the vapour may be constantly inhaled; or the tincture or ointment may be applied externally. Thyroid extract also produces a decided effect on the parenchymatous goîtres in young persons. In any case the treatment may have to be continued for months. Surgical measures may be necessary in very hard or very large goîtres and in malignant disease, if recognised sufficiently early; they are the injection of tincture of iodine, the use of setons, the enucleation of an encapsuled tumour, or the removal of the greater part of the gland.

EXOPHTHALMIC GOÎTRE

(*Graves' Disease. Basedow's Disease*)

This disease was first described by the Dublin physician, Graves, in 1835, and by a German physician, Basedow, in 1840. There are three prominent symptoms: protrusion of the eyeballs, enlargement of the thyroid gland, and frequent action of the heart.

Ætiology.—It occurs much more frequently in women than in men, in the proportion of 11 to 1 (Murray), and mostly between the ages of fifteen and thirty. It has been thought sometimes to follow upon anæmia and chlorosis; but its early symptoms may have been mistaken for one of these. More often there has been a neurotic tendency, as shown in hysteria or epilepsy, or mental disease in the family. In a few cases it has followed rapidly upon some emotional or mental excitement, or even direct injury to the head. A hereditary connection has sometimes been observed: it has been observed in mother and son or daughter; more often it attacks brothers or sisters in the same family.

Symptoms.—With the exceptions just alluded to, it generally comes on quite gradually, and, as a rule, the cardiac symptoms

first appear, the protrusion of the eyeballs and the thyroid swelling not till some months later. Occasionally they may appear in a different order, or one or other of the three chief symptoms may be absent; but the circulatory trouble is the most constant. In the fully developed disease, the *heart* beats quickly and forcibly; the impulse is in the natural position, but it is felt over an abnormally large area; there is often a systolic murmur in the pulmonary area, and occasionally one at the apex. The carotids and large arteries pulsate with great force, and the patient feels the violence both of the cardiac beat and the arterial throbbings. The pulse reaches 120, 130, or 140 in the minute, is of medium size, and yields no special sphygmographic tracing. The patient suffers from shortness of breath on exertion in proportion to the cardiac disturbance. In course of time the heart may become dilated and hypertrophied.

The enlargement of the *thyroid body* is symmetrical, usually moderate in dimensions, and rarely equal to that of the larger endemic goîtres. If the hand be placed over it, a thrill can be felt which is due to the movement of blood in its dilated vessels, and a systolic murmur can be heard with the stethoscope.

The *prominence of the eyeballs* (exophthalmos, proptosis) is the most striking characteristic of the disease, and gives to the patient an unpleasant terrified appearance. It affects both eyes, and may reach such a degree that the sclerotic is seen both above and below the cornea, and the eyelids are unable to meet. Even when the eyelids can be voluntarily closed, they may lie apart during sleep; and if the exophthalmos is extreme, there may be irritation and ulceration of the cornea as a result of exposure. Sometimes the prominence is only simulated in consequence of spasm or retraction of the upper eyelid (Stellwag's sign). In association with the exophthalmos there is a want of uniformity in the movements of the eyeball and the upper lid, so that, when the patient lowers the eyeball to look down, the upper eyelid is not depressed to a corresponding extent (von Graefe's sign). It is not present in every case, although it has sometimes been noticed even before the protrusion. On the other hand, when present it is of importance, as it does not occur in other kinds of exophthalmos. Weakness of the convergent muscles may be also present (Möbius' sign), and in some cases definite paralysis of some or all of the ocular muscles. The pupil and accommodation are unaffected; and the ophthalmoscope reveals little beyond overfull and tortuous retinal veins.

The sufferer from these lesions is out of health in many ways besides. She is languid, anæmic, unfit for exertion, wanting or capricious in appetite, subject to nervousness, headache, vertigo, fits of temper or crying, irritable, or hysterical. In some cases, melancholia, hallucinations, and even mania have occurred; and tetany is an occasional event. There is often a more or less constant *tremor* of the limbs, and even of the whole body. The nervousness and cardiac action are increased by attention or by any excitement.

The patient is generally thin, and may waste a good deal. A moderate degree of fever is often present; and some patients show various pigmentary changes of the skin, such as moderate bronzing, chloasma, or leucodermia. A subjective feeling of heat, flushing of the head and neck, sweating, diarrhœa, glycosuria, and intermittent albuminuria have been noticed in different cases. The electrical resistance of the body is diminished as a result of the moisture of the skin. The voice is often thin, feeble, or piping, sometimes, perhaps, from pressure of the goître on the trachea. The symptoms are liable to aggravation from time to time.

Morbid Anatomy.—In the enlarged thyroid the vesicles, instead of being round or cubical, are branched or stellate, the epithelial cells lining the vesicles become cylindrical instead of cubical, and proliferate into the vesicle; and the contents of the vesicles lose their colloid nature and become mucous and granular. These changes resemble those which occur in compensatory hypertrophy of the remains of the gland after partial removal (Edmunds). In later stages it may become fibrous or cystic.

The thymus gland is often persistent and enlarged, and there is sometimes enlargement of the spleen, of the cervical or bronchial glands, or of Peyer's patches in the intestine.

Pathology.—This is, as yet, very obscure. The protrusion of the eyeballs is probably in part due to venous congestion, and in part to an overgrowth of fat in the orbit. A third factor is believed by some to be present, namely, a contraction of certain non-striated muscular fibres, which have been recognised by Müller, in the membrane lining the orbit over the spheno-maxillary fissure. Such a contraction, it is thought, would force forward the contents of the orbit. The presence of similar smooth muscular fibres in the upper (and lower) lids is considered by von Graefe to explain the symptoms described by him, since they are innervated by the sympathetic. Others ascribe them to weakness of the orbicularis palpebrarum. When it is attempted to explain how these different lesions can be referred to a common origin, considerable difficulty arises. The three cardinal symptoms suggest a lesion of the cervical sympathetic; but nothing has been found in the cervical ganglia or in the brain or spinal cord to explain the symptoms.

The view that this disease is due to excess of an *internal secretion* of the thyroid depends upon the resemblance of the symptoms to those which follow large doses of thyroid extract, and their contrast with those of myxœdema; and improvement has occurred in some cases after partial excision of the hypertrophied gland.

Diagnosis.—Occasionally a *simple goître* may actually press upon the sympathetic in the neck, and produce dilatation of the pupil, paralysis of accommodation, slight exophthalmos, and depression of the temperature in the external meatus. It will be seen that these symptoms agree with those of Graves' disease only in the exophthalmos.

Prognosis.—Many cases are of some years' duration, beginning insidiously, and only slowly recovering; others improve up to a

certain point, and then remain stationary, with but slight prominence of either symptom, or with slight exophthalmos only, or tachycardia alone. These cases altogether amount to 60 or 70 per cent. of the whole; others again, from 15 to 20 per cent. of the whole, die from exhaustion, from cardiac dilatation and mitral regurgitation, from intercurrent complaints, or suddenly, and then probably from heart-failure. In a few cases myxœdema has supervened; Murray saw it only once in 120 cases.

Treatment.—A cure can only be expected from prolonged treatment, and no drug can be said to have been uniformly successful. Improved hygienic conditions, residence at high altitudes, rest or very gentle exercise, and a simple diet are favourable; while tea, coffee, alcohol, and tobacco are likely to be prejudicial. Arsenic, convallaria, strophanthus, digitalis, belladonna, potassium bromide, and ergot are the drugs which have done most good. Sodium salicylate is also recommended. Many cases have benefited under galvanism, the constant current being applied with the kathode on the cervical spine, and the anode over the sympathetic in the neck, or over the thyroid; and a weak current may be similarly applied to the eyes. The faradic current may be used in the same way. Some good results have followed the application of the Röntgen rays for five or ten minutes daily. The application of ice to the thyroid is also of value. Suprarenal extract has been beneficial in some cases: thymus extract has been used with no great success; and thyroid extract is likely to be harmful. The attempt to procure a curative anti-thyroid serum from goats or rabbits after feeding them on thyroid gland or extract has not been successful; but good results have been obtained from feeding patients on the milk of goats, from which the thyroid has been removed; from the use of rodagen, which is a mixture of milk-sugar, and the desiccated milk of goats deprived of their thyroids; and from thyroidectin, which is the dried blood of thyroidectomised animals. The dose of rodagen is a drachm three or four times a day, but it may be increased to twice that amount; and that of thyroidectin is 5 grains given in capsule.

Various operations have been performed on the thyroid body, its arteries, and the sympathetic nerve. But the only procedures that can be recommended, and those only in extreme cases, are partial resection of the gland, and ligature of two or three thyroid arteries.

MYXŒDEMA

(*Cachexia strumipriva*)

Myxœdema and cretinism are the results of disease of the thyroid body, causing deficiency of its secretion (*see* p. 873); cretinism is congenital, myxœdema arises in later life.

The prominent features of myxœdema are a peculiar swelling of the skin and subcutaneous tissue, with dryness and roughness of the surface; pronounced mental failure, consisting of dulness, apathy,

Symptoms.—The onset is generally insidious, and the patient gradually suffers from weakness, depression, languor, and indisposition for exertion. There may be pains in the loins, hypochondrium, or epigastrium, aud tenderness on pressure in one or other hypochondriac region. The heart's action is very weak, and there are faintness and giddiness on rising in bed, or breathlessness and palpitation on exertion. The pulse beats from 80 to 90 in the minute, and is small and feeble: the blood-pressure is very low, measuring as little as 90, 80, or 70 mm. of mercury. Appetite is generally deficient, and nausea, retching, and vomiting are important features of the disease. A peculiar *discoloration of the skin* is the symptom which has attracted most attention, but which it is important to remember may be entirely absent. This symptom may be noticed coincidently with the above general symptoms; it may develop before them, or it may occur several months after they are pronounced. It is presumable, in this last class of cases, that if the general symptoms were very severe they might be fatal before the skin was affected; and thus the occasional absence of pigmentation in Addison's disease of the suprarenal capsules is explained. The pigmentation or *bronzing* is, in its lighter shades, dusky, or yellowish-brown; sometimes of olive or greenish-brown hue. In its more pronounced form the skin has a rich brown colour, like that of a mulatto. The pigment is not scattered or in spots, but more uniformly distributed over different parts of the body—that is to say, large areas are discoloured, the darker tints gradually shading off into the lighter, or into the natural colour of the skin. The change usually invades, first, the parts of the skin which are naturally exposed, such as the face, neck, and the backs of the hands and fingers, but not the scalp or the lip under the moustache; secondly, parts which are naturally more deeply pigmented than others, such as the axillæ, penis, scrotum, and areolæ of the nipples; and thirdly, seats of pressure or slight injury, such as the marks of garters and waistbands in women, and places where blisters and plasters have been applied. But the scars of wounds destroying the skin remain white, and are bordered by a deep layer of pigment. Sometimes there is darkening of the depressed lines in the palms. On the darkened parts of the skin may be seen small black specks, like moles or freckles. In advanced conditions the whole body may be covered by the pigmentation; but as a rule, one must be prepared to recognise the disease, and, indeed, many patients die, before this stage is reached. The pigmentation is not limited to the skin. A bluish-black line may often be seen on the inner side of each lip running along the mucous membrane, parallel to the line of junction with the skin; and other more irregular patches may occur on the mucous membrane of the cheek and on the side of the tongue. Some of these may be determined by the presence and consequent irritation of carious teeth. The temperature is, as a rule, normal; the urine is of medium colour and specific gravity, and free from albumin. Although weak, the patient is not

necessarily emaciated, nor anæmic: even a considerable layer of subcutaneous fat may persist to the end; red corpuscles and hæmoglobin are in normal amount; and there is no leucocytosis.

The course of the disease is very variable. It is often marked by exacerbations and remissions, periods of severe illness, which confine the patient to bed, alternating with times of comparative health; but after each fresh aggravation of the disease the patient is left decidedly worse than he was before it. The duration varies from a few months to six or seven years. Death takes place mostly by asthenia, the patient getting gradually weaker, and lapsing into a drowsy or semi-comatose condition, with increasing feebleness of pulse. Delirium and convulsions occasionally close the scene. In some cases the general symptoms and a very slight pigmentation have been noticed only for some months, when extreme prostration has ensued and carried off the patient in a few weeks.

Anatomical Conditions.—The parts of the body presenting changes are the skin and mucous membrane, the suprarenal capsules and the adjacent parts, and the mucous membrane of the alimentary canal. The change of colour in the former is due to pigment, which is deposited, for the most part, in the deepest layers of the epidermis, a condition similar to that which obtains naturally in the skin of the negro. A few pigment-granules are also scattered in the upper parts of the papillary layer of the true skin.

The suprarenal capsules are usually enlarged, hard, and irregular, and present on section a combination of translucent grayish or greenish-gray tissue, and opaque yellow cheesy substance. There are also conditions intermediate between the two; or the cheesy substance may exist only in the form of nodules in the midst of the other. Small particles or larger masses of cretaceous matter may be found in the caseous substance; or the whole gland may be converted into a cretaceous mass. Sometimes, on the other hand, the caseous matter has softened down into a cavity containing pus. It is obvious that this condition has the closest possible resemblance to a tubercular process, and the identity has been shown by the presence of the tubercle-bacillus in the diseased gland. But another important feature must be noticed—namely, that the chronic inflammation of the suprarenal body leads to thickening of the connective tissue around it and adhesion to neighbouring structures; and that the solar plexus, the semilunar ganglion, and the terminations of the phrenic and pneumogastric nerves are often involved in the disease either by compression or acute inflammation. Enlarged lymphatic glands are also found in the surrounding connective tissue, as well as in the mesentery, and behind the peritoneum. The solitary follicles of the small and large intestines are often swollen, and the mucous membrane of the stomach may be "mammillated" from an overgrowth of lymphoid tissue between the gastric tubules.

In some cases there are found the tubercular changes in the lungs or other parts, or the various abscesses, to which allusion was made when treating of the ætiology of the disease.

In a small number of instances, in which the symptoms of Addison's disease have been undoubtedly present, the capsules have been atrophied to an extreme degree, without evidence of previous tuberculisation.

Pathology.—The accepted explanation of Addison's disease is that the internal secretion of the adrenals is checked or abolished by the pathological change which the organs have undergone. This secretion consists in part at least of adrenalin, which is formed in the chromophile or chromaffin cells of the medulla, and has a powerful influence in maintaining the blood-pressure. Its absence accounts for the low pressure and general asthenia of the disease. The pigmentation and gastro-intestinal disturbances are less easily explained. By some they are referred to implication of the sympathetic in fibrous changes around the gland; and by others to the absence of a possible secretory element in the cortex, by which toxic influences are normally neutralised. But there is much yet to learn about the disease, if only to show how the organ may be destroyed by malignant disease, and even rarely by tuberculo-caseous change, without the usual symptoms of Addison's disease.

Diagnosis.—The mistakes most likely to be made are: (1) to take some other discoloration for that of Addison's disease; (2) to fail in recognising the symptoms when the pigmentation is slight or absent. The discolorations likely to be mistaken for it are slight *jaundice*, which is distinguished by the yellow tinge of the conjunctiva; *phtheiriasis*, to be recognised by the scars, blood-crusts, scratches, pediculi, the limitation of the colour to parts that can be scratched, and the entire freedom of the face; the sallow or earthy tints of *malaria* and of *phthisis; chloasma uterinum* in women; and *tinea versicolor*. In early stages, without much darkening, the apparently causeless weakness, with small feeble pulse and sickness, are the diagnostic features; in some such cases the application of linseed-meal or mustard poultices has been followed by an unusual amount of pigmentation locally. It has been found that suprarenal extract given internally raises the blood-pressure of the sufferer from Addison's disease, but does not affect that of persons in health. The extract should be given in 3-grain doses three times daily for three days, and the blood-pressure should be accurately estimated before and afterwards (O. Grünbaum). The opsonic tests for tubercle may also be applied.

Treatment.—This must be, on the whole, tonic. Sickness must be met by effervescing salines, bismuth, iodine, &c. Iron, arsenic, and strychnine are the most suitable tonics. The internal administration of a suprarenal extract has given some good results, at least temporarily, restoring the strength, but without removing the pigmentation.

ACUTE INADEQUACY OF THE ADRENALS

The adrenals are also subject to acute changes, which are essentially different from those usually found in Addison's disease. In acute infectious diseases, such as diphtheria, there may be cloudy swelling, necrosis, and infiltration with leucocytes. In the same disease hæmorrhages and acute hæmorrhagic necrosis may take place in the medulla, and the same occurs in enteric fever, pneumonia, erysipelas, and purpura. Inflammation and *abscess* from proximity to other suppurating foci; hæmorrhage from injury; *lardaceous* change in common with other organs; miliary tubercles in general tuberculosis; and rarely syphilitic gumma are among the other pathological conditions which may be found.

Some of the acuter infections and hæmorrhagic necrosis are responsible for fatal symptoms, of short duration, which may be reasonably attributed to acute interference with the secretory functions of this gland. Cases of this kind have been classified as follows: Sudden onset of epigastric pain and tenderness, followed by abdominal distension and death within a few days; profound asthenia, ending with death in a few days; cases with convulsions, coma, and delirium, or a typhoid stage; cases of sudden death, in which hæmorrhage is generally found; cases with a purpuric eruption, or with hæmorrhages into the abdominal viscera.

HYPERTROPHY AND ATROPHY OF THE SUPRARENAL CAPSULES

Rare cases occur which show an important relation between the cortex of the adrenals and the nutrition of the body generally. They are those of children from two to eleven years of age, in whom a precocious development of the sexual organs, excessive growth of hair on the body, and obesity have been associated with a hypertrophy of the adrenals, or the existence of the renal tumour which is called hypernephroma, and is believed to be a hyperplasia of the suprarenal remains in the kidney substance (*see* p. 951). Conversely, hypoplasia or atrophy of the adrenals has sometimes been seen in cases of disappearance or original absence of hair from the genitals, and hypoplasia of the genital organs.

TUMOURS OF THE SUPRARENAL CAPSULES

The tumours affecting these glands are adenoma, sarcoma, and carcinoma. Sarcoma occasionally happens in children, and may form a large mass, which may be mistaken for a renal tumour. Carcinoma is rarely primary, but generally a part of widespread secondary lesions.

DISEASES OF THE URINARY ORGANS

EXAMINATION OF THE KIDNEY

The kidney normally extends from the lower border of the eleventh dorsal vertebra to the lower border of the second lumbar vertebra, its inner margin being, on an average, three inches from the middle line; and it is fixed in this position by its surrounding capsule of adipose tissue, by the layer of peritoneum in front of this, and by the renal vessels. It cannot be felt, except in very emaciated persons, or when it is enlarged, or when it is more mobile than normal (*movable kidney*), or when it is pushed down by disease above. Each flank should be examined by the bimanual method, and the patient should be directed to take a deep breath; or in thin persons the flank may be grasped by one hand, the fingers behind and the thumb in front.

A renal tumour may be mistaken for the liver on the right side, or the spleen on the left. If the resonance of the colon is detected in front of the mass, it is not the spleen or the liver.

EXAMINATION OF THE BLADDER

With the aid of the cystoscope valuable information may sometimes be obtained as to the condition of the kidneys. If one or other kidney is the subject of pyelitis, pyelo-nephritis, tubercular disease, or similar lesion, and the ureter is involved, the orifice of the ureter in the bladder may be patent, with swollen lips and vascular adjacent mucous membrane. A purulent or sanguineous fluid may be also seen injected into the bladder from one or other ureter; or the absence of secretion from one kidney may be shown.

EXAMINATION OF THE URINE

QUANTITY

This is on an average fifty ounces, or 1450 cubic centimetres, per diem. It depends directly upon the pressure in the renal glomerulus, and consequently the urine is increased by conditions which raise the arterial tension either generally or in the kidney, and diminished by those which reduce this tension. Excessive secretion

of urine is called *polyuria,* its suppression *anuria.* Daily causes of increase are the ingestion of fluids and exposure of the bodily surface to cold; while it is diminished by abstinence from drink, by free sweating, and the loss of fluid from the circulatory system.

In disease, variations are seen in the following circumstances: The flow of urine is increased in the earlier stages of chronic granular kidney, in lardaceous disease of the kidney, in rare cases of cerebral disease, in diabetes insipidus, and in diabetes mellitus. A temporary increase is seen in hysterical attacks, as a result of mere nervous excitement (not infrequent during medical examination for life insurance), and from the administration of substances having a diuretic action, such as the acetates, citrates, and tartrates, and, perhaps most commonly, alcoholic drinks. The urine is scanty or suppressed in acute nephritis, in the last stages of chronic nephritis, in obstruction of the ureter unless the other kidney is equal to the task of secreting twice its normal amount, in all febrile processes, in cardiac failure, and after repeated vomiting, or profuse diarrhœa. The diminution in fevers is explained by increased transpiration of aqueous vapour through the lungs and skin, and by diminished arterial tension; in cardiac failure the diminished arterial tension is the efficient cause.

In order to ascertain the daily excretion of urine, all that is passed during twenty-four hours should be measured, and the mean of several consecutive days should be taken, so as to exclude the variations which are constantly occurring in health or disease.

SPECIFIC GRAVITY

This varies directly as the amount of solids in the urine, inversely as the quantity of water—that is, of the urine itself. The excretion of both water and solids varies with the period of the day, and it is therefore desirable, for accurate estimation, to mix the whole of the urine secreted during twenty-four hours, and to take the specific gravity, or density, of the combined liquids, which varies between 1000 and 1050, the specific gravity of water being taken as 1000. The *urinometer* consists of a glass bulb, weighted with mercury, and having a vertical stem graduated from 0 to 50. In distilled water it sinks, so that the level of the liquid corresponds to zero; the specific gravity is read as 1000. In different urines it floats at higher levels, and the figure touched by the surface of the liquid, added to 1000, gives the actual specific gravity. If the quantity of urine is too small to float the urinometer it may be diluted with one, two, or three volumes of water, as may be required, and the specific gravity will be obtained by multiplying the last two figures by the number which represents the dilution. Thus, a specimen of urine which, diluted with three volumes of water, gives a specific gravity of 1007, has really a specific gravity of 1028 ($7 \times 4 = 28$). The density varies with the temperature. For accurate record, all observations should be made at 60° F., or

corrections should be made by adding one degree of density for every 8° F. above 60° F.

The urine in health has a specific gravity of 1015 to 1025. It is diminished by most of the causes of polyuria, so as to be 1010, 1006, 1004, or less; as in chronic granular kidney, lardaceous disease, diabetes insipidus, hysteria, nervous excitement, and under the influence of alcohol or other diuretic. It is increased by all the causes of scanty urine, such as heart-disease, acute nephritis, and profuse sweating; and by conditions which increase the solids actually or relatively to the fluid secretion. In diabetes mellitus the specific gravity may be increased to 1030, 1040, or 1050, although the quantity of urine is many times more than the normal; the unnatural secretion of large quantities of sugar, while urea is often above the normal, accounts for this exceptional condition. Albumin occurs in urine of both low and high specific gravity; it must help in increasing the density, but it is, as a rule, in very small quantity when the conditions of the kidney allow a free secretion of urine.

SOLIDS

The daily average of solids amounts to 950 grains, or 58 grammes. Their amount is accurately estimated by evaporating a specimen of the urine (collected during twenty-four hours), and weighing the residue. Since the specific gravity is in proportion to the solids, it ought to be possible to calculate the latter from the former with some degree of correctness. The average yield of solids on evaporation is about 4 per cent.; the average specific gravity is 1020. If the last two digits, 20, are multiplied by 2 we get 40—that is, the amount per 1000, and this corresponds to 4 per cent. Hence the rule to multiply the last two digits of the specific gravity by 2, in order to obtain the amount of solids per 1000, or grammes per litre; they must then be multiplied into the figure representing the quantity of urine, and divided by 1000; thus:

$$\text{(Daily amount in cub. cent.)}\ \frac{1450 \times 40\ (=20 \times 2)}{1000} = 58 \text{ grammes.}$$

The figure 2 thus employed is called *Trapp's co-efficient*, but the figure 2·33—*Haeser's co-efficient*—is thought to be more accurate, especially for specific gravities over 1025. The constituents of the urine whose increase is likely to affect the total daily solids are urea; the chlorides, phosphates, and sulphates of sodium, potassium, ammonium, calcium, and magnesium; urates of sodium, ammonium, and calcium; and the abnormal constituents—sugar and albumin. A great increase in solids is found in pyrexia. Excess of the nitrogenous constituents (urea) is called *azoturia;* the presence of sugar and albumin respectively *glycosuria* and *albuminuria.* There is an actual diminution of solids in the conditions known as *renal inadequacy* and *anazoturia.*

The following require further consideration:

Urea.—This constitutes about one-half the total solids of the urine, and is the chief form in which the nitrogen of the body is excreted. Its presence in the urine can be shown by evaporating an ounce of urine to about one-third its bulk, and adding a drachm of strong nitric acid: a flaky or feathery mass of crystals of nitrate of urea will be deposited as the mixture cools. If the urea is very abundant, or the urine is highly concentrated, this precipitation will occur without previous evaporation.

Quantitative Estimation.—A convenient method of estimating the urea is to decompose it by the addition of *sodium hypobromite* (or *hypochlorite*) into carbonic acid, water, and nitrogen—

$$(CON_2H_4 + 3NaBrO = 3NaBr + CO_2 + 2H_2O + 2N).$$

The nitrogen is measured, and from that the amount of urea can be calculated, the proportion of urea to its contained nitrogen being as 60 to 28, or 15 to 7. Really the hypobromite method shows only about 92 per cent. of the nitrogen contained in the urea, but it obtains another 2 per cent. from creatinin and other bodies.

There are several forms of apparatus (Russell and West, Southall, Doremus, Granville); in all of these a measured quantity of urine (5 c.c.) is mixed with a quantity of the hypobromite solution in such a way that the nitrogen gas, which immediately comes off, may be collected and measured in a graduated glass tube. The other results of the decomposition remain in solution. The graduations may represent the actual volume of nitrogen. In the apparatus generally employed the graduations represent not the volume of nitrogen gas, but the percentage of urea in the urine to which such volume is equivalent, and a tedious calculation is thus saved. The hypobromite solution rather readily undergoes decomposition after keeping, and it is better to make it afresh for each analysis.

In *Squibb's process* the liquor sodæ chloratæ of the U.S. Pharmacopœia, containing sodium hypochlorite, is used, and the nitrogen displaces water from a bottle completely filled and connected by a tube with the vessel in which the decomposition takes place. The displaced water has the same bulk as the liberated nitrogen, and the calculation of the urea can be made therefrom.

The average daily excretion of urea is about 500 grains, or 32 grammes; it forms normally about 2 per cent. of the urine. The quantity varies very much, and is influenced most by the amount of nitrogenous food taken, being increased or diminished in proportion. It is increased also by the ingestion of water, and of table or other salts, and by the addition of fat to nitrogenous diet; but the further addition of farinaceous food again diminishes it. Moderate muscular exercise does not affect it.

In relation to disease one must not rely only upon the percentage, but upon the absolute daily amount as far as that can be ascertained. The percentage may be high in consequence of diminished passage of water, as after sweating, or restricted supply of water, while the daily excretion of urea is normal. An increase of urea takes place

in fevers, and in diabetes. There is a considerable decrease in all forms of Bright's disease, but also in other diseases, in which the amount of food ingested is much less than normal.

Chlorides.—Their presence is shown by a white precipitate on the addition of nitrate of silver to the urine, acidulated with nitric acid. Normally, the precipitate is thick and curdy: with diminished chlorides it may be a mere turbidity. The daily excretion of chlorine is about 7 grammes, equal to about 15 grammes of sodium chloride. The chlorides are lessened in all acute febrile processes, and especially in pneumonia, where they may be entirely absent.

Sulphates.—The daily excretion of sulphates is from 1·5 to 3 grammes. Of these the greater part are alkaline sulphates, precipitable by barium chloride; but from 5 to 10 per cent. are aromatic or ethereal sulphates, not precipitable by barium chloride, but convertible into the alkaline sulphates by boiling with hydrochloric acid. The ethereal sulphates are partly the result of putrefaction in the intestines, and are largely increased in disorders of the intestines (obstruction, peritonitis, constipation) leading to such changes.

Phosphates.—The daily excretion of phosphoric acid in the urine is from 2 to 3·5 grammes, combined with the alkaline bases, sodium and potassium, and with the earthy bases, calcium and magnesium. The amount is chiefly determined by the food, phosphoric acid being provided by tissues rich in nuclein. The *alkaline* phosphates form about two-thirds of the whole, and are always held in solution; the *earthy* phosphates form about one-third, and are held in solution as long as the urine is acid; but they are precipitated from the urine in various circumstances, as phosphatic deposits and calculi.

The common cause of such deposition is an alkaline condition of the urine. If the urine becomes alkaline in the body, or after it has been passed, phosphates are deposited. The urine in health may be temporarily alkaline from the ingestion of much vegetable matter or other food containing citrates, tartrates, or malates of potassium and sodium. These are converted into alkaline carbonates in the intestines, and absorbed as such into the blood, and hence diminish the acidity of the urine, or render it alkaline.

Occasionally the urine, when passed, is quite turbid from a white deposit, consisting mainly of amorphous phosphate of calcium ($Ca_3P_2O_8$). This settles as a bright white deposit, which is immediately soluble in dilute acetic or nitric acid. Rarely there is mixed with this a crystalline phosphate ($CaHPO_4 + 2H_2O$), showing under the microscope rods and needles, lying loose or grouped into rosettes, stars, fans, and sheaf-like bundles, or club-shaped or bottle-shaped masses. This is called sometimes stellar phosphate. A magnesian phosphate ($Mg_3P_2O_8$), in elongated plates with oblique ends, has also been identified.

A much more common occurrence is this, that on heating a feebly acid or neutral urine a turbidity or thick white deposit of earthy phosphates occurs, which closely resembles albumin, but is at once distinguished from it by being dissolved by a drop of acetic or nitric acid. The heat acts by decomposing the dicalcic phosphate ($Ca_2H_2P_2O_8$), held in solution by other salts, into a monocalcic phosphate ($CaH_4P_2O_8$) and the tricalcic phosphate ($Ca_3P_2O_8$), which is insoluble. The addition of liquor potassæ or liquor sodæ to the normal urine at once throws down the phosphates.

The urine also becomes alkaline from the presence of ammonia, which is formed as the result of bacterial decomposition of urea, either within the bladder (cystitis) or after standing in a vessel exposed to the air. In this case the *ammonio-magnesian* phosphate is precipitated ($MgNH_4PO_4+6H_2O$). It forms triangular prisms, with bevelled ends, often very perfect indeed, but sometimes modified by the edges or angles being, as it were, planed off, or the ends or surfaces hollowed out. They often reach a large size, and are strongly refracting. This deposit may be mixed with that of the phosphate of calcium.

Calcium Oxalate.—The quantity of calcium oxalate secreted daily is very small, but it is sometimes seen as a deposit in the urine, or it forms calculi in the kidney. In urine containing an oxalate deposit there is mostly a pale gray mucus-like sediment, and above this a white dense layer, with a wavy, sharply defined surface. Under the microscope will be found the minute octahedra of calcium oxalate, looking often like square envelopes, and measuring from $\frac{1}{5000}$ to $\frac{1}{750}$ inch in the side. In different positions they may seem to have a rhombic or hexagonal outline, and if the edges are not developed they may form square prisms, with pyramidal ends. A not uncommon variety is that of the "dumb-bell," which is really a disc, with a central depression on either face, lying on its side, and seen edgeways. Such formations no doubt result from slow precipitation in the presence of colloid matter.

Calcium oxalate crystals are with difficulty soluble in hydrochloric acid, and insoluble in acetic acid. Their deposition is sometimes due to excessive ingestion of vegetables (cabbage, rhubarb) containing oxalates; or to changes in the urine after secretion; or to disturbances of digestion. Their connection with symptoms such as languor, depression, and hypochondriacal feelings (the so-called *oxalic acid diathesis*) is very doubtful, unless they and the symptoms together are the results of indigestion.

Uric Acid and Urates.—The daily excretion of uric acid, which is in combination as urates, is only from 8 to 15 grains, or 1 gramme, so that the proportion in the urine is very small; nevertheless, deposits of the free acid and of its salts are not infrequent. As in the case of the other constituents, this precipitation does not prove that the quantity actually formed is in excess.

Uric acid is generally precipitated in very acid urine. Its deposits are distinguished by their yellow, orange, or red colour,

and consist of minute shining grains, which with a lens or even the naked eye can be seen to have a crystalline, acicular, or prismatic shape. Under the microscope they show themselves as fusiform or lozenge-shaped crystals, with sharp ends and rounded sides; or as shorter and thicker crystals, with blunt extremities and more barrel-shaped; but generally in some modification of the diamond shape. They are frequently grouped together in radiating clumps or star-like masses. Thick crystals lying on their edges may seem to be rectangular, and the lozenge shape may not be detected till they roll over. Sometimes the sides of such masses are marked by a division into several plates (*striated*). These differences of shape are due to the conditions of the medium in which precipitation takes place. Their colour is that of the urinary pigment, for which they have great affinity. The crystals are insoluble in acids, but can be dissolved in alkalies.

Urates are, as a rule, precipitated, in an amorphous form, as a thick pink or red sediment (*brick-dust* or *lateritious*). The causes of their deposition are (1) the cooling of the urine, because they are abundantly soluble at the body-temperature, and very much less so at 50° or 60° F.; and (2) the concentration of the urine, or the relative deficiency of the water, so that when cooling takes place it is insufficient to keep in solution the perfectly normal amount of urates present. The usual causes of concentration are, in health, deficient intake of fluids, and free perspiration; in disease, vomiting, cardiac disease which acts by lowering arterial pressure in the renal circulation, and febrile reaction causing diminished arterial tension, and increased cutaneous and pulmonary transpiration of aqueous vapour. A gentle heat will at once dissolve the deposit —for instance, the addition of hot water to the vessel containing it; and if the urine is being tested for albumin, the urates dissolve, and leave the fluid clear before the albumin begins to appear. This deposit consists chemically of the urates of sodium, potassium, ammonium, and calcium, which are acid salts, but whether *quadrurates* (formula MHU,M_2U) or *biurates* (MHU) is open to question. *Ammonium biurate* occurs in ammoniacal urines in the form of dumb-bells or spheres, and the deposit is generally pale: *sodium biurate* constitutes the well-known acicular crystals found in gouty joints. *Neutral urates* (M_2U) do not occur in the animal body. The colour is due to a urinary pigment (*uroerythrin*), but it may be absent or amount to nothing more than a yellow tinge.

The Murexide Test.—If a particle of uric acid or a urate is heated on porcelain or a glass slide with a drop of nitric acid and a drop of ammonia and water is added to the dry residue, a purple-red colour is developed. Liquor potassæ changes this to purple-blue.

The *quantitative estimation* of uric acid in the urine presents many difficulties, and is not suited for ordinary clinical work. A rough method is to add to a measured quantity of the urine about one-twentieth of its bulk of strong hydrochloric acid. The uric acid precipitated after twenty-four hours is collected, dried, and weighed;

but the separation of the uric acid is incomplete, and the estimate is consequently too low.

Uric acid ($C_5N_4H_4O_3$) is one of the purin-bodies, that is, substances containing the nucleus (C_5N_4); and these are derived partly from the food ingested and partly from the metabolism of tissues in the body, such as muscle and the nuclei of breaking-down cells. It has an important relation to gout; and it is excreted in very large quantities in some diseases, *e.g.*, leuchæmia. Other purin-bodies are xanthin, hypoxanthin, adenin, and guanin.

Urinary Pigments.—The urine varies in colour both in health and disease. Vogel has prepared and published a series of tints, varying from a very pale tint of yellow to red, red-brown, and almost black, and specimens of urine may in particular cases be referred to these for comparison. But, as a rule, it is sufficient to distinguish between pale urines, normal-coloured urines, and high-coloured urines in health; while in disease we may observe, in addition, different shades of red, reddish-brown, and brownish-black from the admixture of blood or bile-pigment, and an opaque white colour in chyluria.

The difference of tint in pale and high-coloured urines is mostly dependent upon the amount of water contained in them. *Pale* urines are of low density, and contain a large quantity of water and a small percentage of solids. They result from all causes which increase the flow of urine, such as free ingestion of fluids; a check to the cutaneous transpiration, as from cold; increased arterial pressure, as in early chronic Bright's disease; and nervous conditions, as hysteria, nervous excitement, and diabetes insipidus. The urine of diabetes mellitus forms an exception in being pale and abundant, while it has a very high density from the quantity of sugar contained in it. *High-coloured* urines are commonly of great density, scanty in quality, and contain a large percentage of solids. They occur after profuse sweating, or diarrhœa; in fever, and in disorders of the circulation, by which the flow through the glomeruli is diminished.

There are several pigments in the urine. Probably its colour is chiefly due, as shown by A. E. Garrod, to an iron-free pigment named *urochrome*. This pigment obscures the violet end of the spectrum, but gives no absorption bands. *Urobilin* exists in the normal urine in only small quantities: 1 to 2 grains in twenty-four hours; it gives a definite spectrum, with an absorption band at Frauenhofer's line F—that is, between the green and the blue. The chemical test of its presence is to render the urine strongly alkaline with ammonia solution, filter, and add a few drops of a 10 per cent. aqueous or alcoholic solution of zinc chloride: a green fluorescence occurs if urobilin is present. Urobilin is probably absorbed from the intestine, where it is originally derived from bilirubin. Urobilin is in excess in fevers, in some diseases of the liver, in excessive hæmolysis (*e.g.*, in pernicious anæmia), and during absorption of extravasated blood. It is diminished in chlorosis, and when the formation of bile is

checked (phosphorus poisoning, acute yellow atrophy) or the bile duct is occluded. *Hæmatoporphyrin* is derived from hæmoglobin; it occurs in minute quantities both in health and in disease, and more abundantly in the urine of rheumatic fever and some other disorders. It may not cause any appreciable difference in tint, and though it exists in quantity in some dark red urines passed after the administration of sulphonal, the dark colour is mainly due to other pigments. *Uroerythrin* is another pigment, which gives the colour to pink urates. Uric acid deposits are coloured by urochrome, and also sometimes by uroerythrin (Garrod).

The urine may also contain *chromogens*—that is, bodies which do not at the time colour the urine, but develop a colour, either on standing, or on the addition of oxidising agents. The following are known: (1) The chromogen of urobilin, urobilinogen, shown to exist in febrile urine, by the addition of nitric acid; (2) a chromogen found sometimes in the urine of anæmia, which, though quite pale when passed, may yield a deep red colour on the addition of nitric acid; (3) the chromogen of *melanin*, a black pigment which is developed on exposure, or on the addition of nitric acid, in the urine of patients suffering from melanotic sarcoma, although it is clear when passed; (4) indican, or potassium indoxyl-sulphate, the chromogen of indigo-blue. This is the result of the absorption from the intestinal canal of indol, which results from the bacterial decomposition of proteids. It exists in normal urine to a very small extent, but is greatly increased in all conditions leading to retention of intestinal contents, such as constipation, intestinal obstruction, and peritonitis. Its presence is detected by the addition of an equal quantity of hydrochloric acid, and then a few drops of a saturated solution of oxychloride of calcium. Indigo is thus formed, and colours the mixture blue or violet. It can be separated by shaking with chloroform, which then forms a blue layer at the bottom of the test-tube. Sometimes the addition of nitric acid alone develops a blue, violet, or blackish colour, due to the separation of indigo.

In *alkaptonuria* the urine is of a natural colour when passed, but darkens on exposure, is darkened rapidly by alkalies when warmed, is turned deep blue by a dilute solution of ferric chloride, and is found to contain homogentisic acid, and sometimes uroleucic acid. The condition is rare, but in many of the cases it is congenital, and occurs in two or more members of the same family, and the children of parents who are blood-relatives. It causes no symptoms (*see* Ochronosis).

Several *medicinal and other chemical substances* colour the urine, or give colour-reactions with tests employed for other purposes. Rhubarb, which contains chrysophanic acid, makes the urine a deeper yellow; and santonin the same. The addition of an alkali will turn these urines red. Logwood gives a reddish tinge to the urine. Carbolic acid, taken internally, or absorbed from carbolic dressings, often causes the urine to become dark-brown or

greenish-black on exposure, though clear when passed, from the presence of pyrocatechin and hydrochinone. Creosote may have the same effect. Methylene blue, taken internally, renders the urine blue, or, if in small quantity, green. Eosin, used to colour toys or sweets, has caused a bright red urine in children. If potassium iodide or potassium bromide is being taken, nitric acid may darken the urine from the liberation of free iodine or bromine. Free iodine or bromine can be separated by shaking with chloroform.

REACTION OF THE URINE

The urine is, as a rule, acid in reaction, and this acidity is probably due to the presence of the acid phosphate of sodium. After a meal the urine is often alkaline. The acidity of the urine during twenty-four hours is equivalent to about 14 grains of carbonate of sodium, or to 30 grains of oxalic acid. After it has passed from the body the reaction of the urine generally undergoes a change, becoming first more acid, then again less acid, and finally alkaline. These changes are due to fermentation from the presence of micro-organisms; for if urine is properly protected from contact with such bodies, it may be preserved for years. The increased acidity is due to more acid phosphates, as well as lactic and acetic acids. The alkalinity results from the decomposition of urea and the formation of ammonium carbonate. In the former case uric acid is often deposited; in the latter, ammonio-magnesian phosphates, ammonium urate, and calcium phosphates and carbonate are thrown down.

It is, therefore, always desirable to know the reaction of the urine immediately it is passed from the body. If it is then alkaline, it must be ascertained whether the alkalinity is due to the volatile alkali, ammonia, or to fixed alkali (alkaline phosphate of sodium). A piece of red litmus paper turned blue by the urine, and subsequently gently warmed over a spirit lamp, will lose its blue colour in the case of ammonia, but will retain it in the case of the fixed alkalies. If ammonia is shown to be present, it is due to decomposition of the urine from retention in the bladder or other parts of the urinary apparatus, as in pyelitis and pyonephrosis, precisely similar to what takes place in ordinary urine after it has been passed some time and exposed to the air.

If the alkalinity is due to fixed alkalies, this results from changes in the blood, whether from diminished supply of acids, or increased supply of alkaline constituents. The most certain means of producing alkalinity of the urine is by the ingestion of large quantities of the potassium or sodium salt of citric, tartaric, acetic, or malic acid. These organic acids are decomposed in the intestines, and the alkalies are secreted in combination with the feeble acid, carbonic acid. The alkalinity which follows a meal is due partly to diversion of acid to the gastric juice, and partly to the ingestion of alkaline citrates, &c., in vegetable food. Probably the latter has most influence, since, after a highly flesh or milk diet, the acidity is usually

increased. The acidity of the urine is often diminished in melancholia, paralysis, anæmia, and chlorosis. It is increased in fevers and diabetes.

DISEASES OF THE KIDNEYS

NEPHRITIS AND BRIGHT'S DISEASE

GENERAL CONSIDERATIONS

To Dr. Richard Bright belongs the credit of having first recognised the association of general dropsy and albuminous urine with a morbid condition of the kidneys. He found the kidneys sometimes large, pale, and smooth; at others, small, dark, and granular on the surface; and he regarded these two forms as different stages of the same process —namely, the deposit of a material which subsequently contracted. However, these changes are largely of an inflammatory nature, and thus *Bright's Disease* came to be almost synonymous with non-suppurative inflammation of the kidney. But a closer study of kidney diseases shows that nephritis may arise in many conditions, which do not fall within the range of Bright's diseases. Thus nephritis is caused by: (1) the toxins of various diseases, such as scarlatina, measles, and diphtheria; (2) micro-organisms carried by the blood-vessels as seen in the metastatic suppurative nephritis of pyæmia; probably also in pneumococcal nephritis, typhoid nephritis, the speckled kidney of malignant endocarditis, and the more local effects of embolism in the same disorder; (3) micro-organisms spreading up the urinary passages, as in so-called consecutive suppurative nephritis (ascending nephritis), in tubercular nephritis, and in nephritis from infection with bacillus coli communis; (4) the presence of calculus in the pelvis of the kidney; (5) alcohol, lead, and gout have a prejudicial effect on the kidney; and though these effects are often degenerative in kind rather than inflammatory, it is impossible to deny that inflammation has sometimes a share: it is admitted that alcohol has a powerful predisposing influence in the production of nephritis from other causes, and possibly it is itself the cause sometimes; (6) the toxins of syphilis and of the pyogenetic micro-organisms produce in the kidney the lardaceous degeneration; this is frequently accompanied by inflammatory changes both in the epithelial and connective tissues, so that the toxins concerned are probably the cause of the inflammation as well as of the degeneration; (7) the red granular kidney is generally regarded as a degenerative condition, resulting from age, or from the influence of the poisons of alcohol, gout, or lead; and the inflammation, of which the evidence (cell-infiltration) can scarcely be denied, may perhaps rightly be looked upon as secondary.

From the histological standpoint one must recognise the following

structures in the kidney: (1) The tubules with their epithelium, forming the parenchyma of the kidney; (2) the interstitial tissue, very small in quantity in the healthy organ, but liable to considerable increase by inflammatory processes; (3) the blood-vessels, and the glomeruli, consisting of the vascular tuft, the capsule, and the epithelial cells covering the former and lining the latter. These tissues are somewhat differently involved in various diseases of the kidney. Thus in some forms of inflammation the epithelium of the tubules is chiefly involved: *tubal, desquamative,* or *parenchymatous* nephritis; in others, the connective tissue: *interstitial* nephritis; of these the chronic cases go on to fibroid contraction: *cirrhotic* or *granular* kidney. In other acute cases the vascular tufts are most affected: *glomerular* nephritis. The vessels as well as the glomeruli are early invaded by the lardaceous degeneration: *waxy, amyloid,* or *lardaceous* kidney; and the small arteries are sometimes thickened and sclerotic: *arterio-sclerotic* and *granular* kidney.

But these distinctions are not absolute: in tubal nephritis the connective tissue does not escape, and conversely, in the granular kidney the tubules are involved; moreover, the lardaceous disease is accompanied by both tubal and interstitial changes.

It is thus better to regard nephritis as in nearly every instance a diffuse inflammation, affecting the several tissues at the same time, but in different circumstances involving the tubal epithelium, or the glomeruli, or the intertubal tissue most, so that a distinction between tubal, glomerular, and interstitial kinds may still be maintained. In addition, we must recognise both *acute* and *chronic* lesions.

Among the above forms of nephritis, the term *Bright's disease* is generally applied to:

1. *Acute nephritis* in part—*i.e.*, when arising from fever toxins, or without obvious cause, and especially when accompanied by dropsy. The inflammation may be tubal, glomerular, or interstitial.
2. *Chronic tubal nephritis,* producing a large white kidney.
3. *Chronic interstitial nephritis,* resulting in a contracted kidney.
4. The red granular kidney with predominant arterial changes is by some regarded as not being inflammatory, and therefore not to be included under Bright's disease. But there can be little doubt that Bright included these cases in his observations; and the cases during life are commonly still so classed.

The lardaceous kidney is at the present time by common consent excluded from the category of Bright's diseases, unless the degeneration should be grafted upon a preceding tubal inflammation.

There are some symptoms which occur more or less in all forms of Bright's disease, and which will now be described. These are—(1) Albuminuria. (2) Hæmaturia. (3) The presence of casts in the urine. (4) Dropsy. (5) Cardio-vascular changes. (6) Ocular changes. (7) Hæmorrhages. (8) Secondary inflammations, and other lesions. (9) Uræmia. (10) Inadequacy of the renal functions.

ALBUMINURIA

The presence of albumin in the urine is the most constant sign of Bright's disease, and may be detected in various ways.

Heat.—If clear acid urine containing albumin be heated in a test-tube, it will become opaque from the precipitation of this substance. According to its quantity the precipitate will be a mere opalescence, a decided turbidity, or a thick creamy deposit; on the cooling of the urine it will separate into small particles or flakes, and gradually subside to the bottom of the test-tube. The best way of applying the test is to fill a test-tube to one-half or two-thirds of its length, and, holding it by its lower end, to warm the upper part of the urine. The heat is thus confined to that portion of the urine, and whatever slight change takes place in the clearness can be recognised by comparison with the cool urine below; whereas, if heat were applied to the bottom of the test-tube, it would reach, by convection, the whole of the urine at once, and a slight opalescence might, for want of comparison, be overlooked. The value of this method is shown in cases where an albuminous urine is turbid from a deposit of urates. Heat will at first dissolve the urates, and then precipitate the albumin. A long column of such urine may be heated in its upper two-thirds to clear it from urates, and then further heated in its upper third to throw down the albumin, when the three strata of urates, clear urine, and albumin may be compared with one another.

Some precautions are necessary. First, heat may precipitate substances which are not albumin. In certain states of the urine, a precipitate comes down which is to the eye indistinguishable from albumin; it is due to the earthy phosphates (*see* p. 891), and is at once dissolved by a drop of nitric or acetic acid, whereas a precipitate of albumin is unaffected or becomes denser. If the two occur together, there will only be a partial clearance on the addition of nitric acid. Heat also throws down serum-globulin, or paraglobulin, which probably always accompanies the serum-albumin of Bright's disease.

Secondly, albumin, though present, may fail to be coagulated by heat. This occurs when the urine is alkaline, and the serum-albumin has been converted into alkali-albumin, which is not precipitated by heat; the fallacy can be guarded against by the addition of a drop or two of acetic acid to the urine before boiling, so as to render it faintly acid. On the other hand it occasionally happens that serum-albumin is converted into acid-albumin or syntonin, which is similarly unaffected by heat. This may happen if the urine boiled is placed in an unwashed test-tube containing a trace of nitric acid from a previous examination. In any case the experiment must be performed upon a clear urine: if turbid from urates, a gentle heat clears it; if from phosphates, a drop or two of acetic acid should be added; if from any other deposit, the urine should be filtered.

Nitric Acid.—This precipitates serum-albumin, as well as alkali-albumin, and acid-albumin. If the albumin is in large quantity, a drop or two of strong nitric acid added to the urine will carry down a thick curdy white precipitate; but for smaller quantities, the test is best applied by placing a little nitric acid in the bottom of a test-tube, and very gently pouring the suspected urine down the side of the tube, so that it flows on to the surface of the acid without mixing with it. At the line of junction a layer of albumin forms, of white colour if abundant, or a thin gray disc when the quantity is very small. In the former case also it forms at once, in the latter it may take several seconds, or two or three minutes, or even half an hour.

There are but few fallacies connected with this test. First, in urine containing an excess of urates, these are sometimes precipitated as a cloud or haze some distance above the nitric acid, instead of lying immediately upon it; the application of a gentle heat will at once dissolve them. Secondly, nucleo-protein (mucin) is precipitated as a haze in the same position. Thirdly, the urine of persons who are taking copaiba internally contains a resinous acid, copaivic acid, in combination with bases. If nitric acid be added to this the resinous body is thrown down, generally as a cloud, evenly diffused through the urine. This precipitate is also dissolved by heat. Fourthly, in many specimens of urine the addition of nitric acid gives rise after some time, when the mixture has become cold, to a crystalline deposit of nitrate of urea; but it has no resemblance to albumin, consisting, as it does, of lamellar crystals, radiating in various directions. Nitric acid also precipitates albumoses, which are redissolved by heat (*see* p. 904).

Picric Acid.—A test-tube should be more than half filled with urine, and a saturated solution of picric acid, which has a low specific gravity, should be poured on to it so that the liquids may mix as little as possible; at the line of junction a delicate white line, or a thicker white cloud, at once forms, which, if albumin, is not dissolved by heat. Besides albumin, picric acid also precipitates urates, alkaloids, and albumoses. These are said to disappear at once on warming the urine. Quinine is the only alkaloid that is likely to be taken in sufficient quantity to be precipitated by picric acid. Nucleo-protein is also precipitated by picric acid and is not dissolved by heat. If picric acid gives no precipitate, albumin is certainly absent.

Salicyl-sulphonic Acid.—A saturated solution of this acid is a delicate test for albumin: added to clear urine in a test-tube, it throws down an opalescent cloud of albumin. Albumoses are also precipitated.

Potassium Ferrocyanide.—This reagent throws down albumin in acid solutions. Citric acid is therefore used as well as the ferrocyanide. The two reagents may be used in solution or in the form of the "pellets" devised by Pavy, which may be easily

carried in the pocket. Into the suspected urine a citric acid pellet is first dropped, and when that is dissolved, a pellet of the ferrocyanide; if any albumin is present it forms a cloud as the salt dissolves. Citric acid itself may precipitate urates and nucleo-protein; if the former are thrown down, a fresh specimen of the urine should be diluted with an equal quantity of water and tested again. If nucleo-protein is precipitated, this specimen should be left untouched, and to another specimen both citric acid and ferrocyanide should be added, when the difference between the two deposits will show the presence of albumin. The ferrocyanide also throws down nucleo-protein and albumoses.

Brine.—A saturated solution of common salt, acidulated with 1 per cent. of hydrochloric acid, may be applied in the same way as nitric acid, by placing about a drachm in the bottom of the test-tube, and pouring the urine gently on to it; the albumin is precipitated at the line of junction. Brine also precipitates albumoses.

Magnesium Sulphate.—A saturated solution of Epsom salts, similarly acidulated, is another useful test.

The following are *quantitative tests* for albumin:

Weight of Precipitate.—The most accurate method is to precipitate by heat and alcohol all the albumin from a definite quantity of urine, and then wash, dry, and weigh the precipitate. This is a troublesome process, and is not suitable for ordinary clinical purposes.

Measurement of Precipitate.—The urine is boiled in a test-tube, the albumin is allowed to settle at the bottom, and after complete subsidence the quantity is compared with the total amount of urine used; it may be one-tenth, one-third, one-half, or more. This gives no information as to the absolute amount of albumin in the urine, but it may show the daily variations of the albumin in any case, and whether the urine contains much or little of it. A very albuminous urine becomes almost solid on boiling; a large quantity settles down to one-half or one-third of the urine in the tube; a mere turbidity, on boiling, will form a little deposit at the bottom of the tube—that is, perhaps from a hundredth to a fiftieth part.

Esbach's Test.—In this the same principle is applied somewhat differently. A test solution is prepared, consisting of one part of picric acid and two parts of air-dried citric acid in one hundred parts of water. A graduated tube from six to eight inches long and a half inch in diameter is filled up to a certain level (two and a quarter inches) with urine, and then for a certain distance (one and a half inches) with the picric solution. The precipitated albumin is allowed to settle for twenty-four hours, and then the space it occupies in the tube is compared with a series of marks which represent the number of grammes of albumin for each litre of urine; or, in other words, the amount of albumin per thousand of urine. Thus, for instance, if it reaches mark 3, there are three grammes of albumin for each litre of urine, or 3 per 1000, or ·3 per

cent. From this, of course, if the daily amount of urine is known, the absolute quantity of albumin passed can be calculated. In some urines the deposit is increased by the presence of uric acid, and thus the albumin may be over-estimated. The test is probably not very exact, but is decidedly useful for comparative estimates.

Roberts' Test.—This consists in a delicate and special application of the nitric acid test (*see* p. 900). Roberts found, when the amount of albumin was so small as not to be visible until from thirty to forty-five seconds have elapsed, that it proves on careful chemical analysis to be equal to ·0034 per cent. If the urine contains more than this the specimen must be diluted—by successive equal quantities of water—until the albumin is in sufficiently small quantity to require the time named. The amount of albumin is obviously directly proportional to the number of times the urine has been diluted by its own quantity of water. Thus, a urine requiring to be increased to ten times its bulk must contain ten times the amount of albumin specified—*i.e.*, 10 × ·0034 per cent. It will be seen that if thirty dilutions are required, the albumin will amount to ·1 per cent. or 1 gramme per litre.

The Causes of Albuminuria.—In considering the reason why albumin appears in the urine in Bright's disease, we must remember that the occurrence of albuminuria is not limited to cases of nephritis, but accompanies a variety of other disorders. The different conditions in which albuminuria has been observed may be enumerated as follows:

I. Arising in the kidney—

1. Acute and chronic nephritis, and contracted kidney, forming Bright's diseases; consecutive nephritis and cystic kidney.
2. Suppurative nephritis.
3. Degenerative changes, such as lardaceous disease and tuberculous kidney.
4. Acute febrile processes, probably causing temporary degeneration of the renal cells.
5. Venous obstruction in diseases of the heart and lungs, and local disturbances of the circulation.
6. Malignant endocarditis and embolism of renal arteries.
7. New growths and parasites.
8. Temporary obstruction of the ureters.
9. Nervous disorders such as apoplexy, convulsions, and concussion.
10. Chronic general disorders, like leuchæmia, diabetes, and anæmia.
11. Disturbances of digestion, and disorders of a temporary nature, such as that caused by violent exertion.
12. Albuminuria of adolescence (*see* Functional Albuminuria).
13. The influence of certain poisons, and the presence in the blood of forms of albumin other than serum-albumin.

II. Arising in the urinary passages below the kidney—

1. Disease of the pelvis of the kidney, calculous pyelitis, and tubercular disease.
2. Tubercular disease of the ureter.
3. Cystitis.

In this last group there is no difficulty in explaining the occurrence of albumin; it is not infrequently accompanied by blood, and may be the result of rupture of vessels, as in the case of calculus, or it is due to inflammatory and ulcerative processes by which an albuminous secretion is provided in the urinary passages, just as it would be on any other mucous surface or on the skin.

The cause of renal albuminuria has been the subject of much debate, but it is generally believed now that it depends upon damage to the renal epithelium; especially that lining the glomeruli. It will be seen that this explanation is more or less applicable to a great number of the conditions under which albuminuria arises.

In acute and chronic forms of nephritis glomerular changes are almost constant, and according to Ribbert they are always the earliest indications of inflammation. In febrile processes, the epithelium of the tubules has long been known to be affected with the condition known as cloudy swelling, and the still more delicate glomerular epithelium would almost certainly not escape. In the venous obstruction of heart disease, it is quite likely that the epithelium suffers from the deficient supply of blood through the afferent arteries. This is supported by Nussbaum's experiment, in which a temporary ligature of the renal artery was followed by albuminuria on restoration of the arterial flow; presumably in the interval the glomerular epithelium was sufficiently damaged to be unable for a time to resist the passage of albumin. In nervous diseases there may be vasomotor disturbances which would act in like manner upon the glomerular circulation; and in chronic disorders the glomerular epithelium may share in the general depression of vitality.

But though this may be the mechanism of albuminuria in a great number of instances, it seems difficult to believe that at no time can it result from transudation directly into the tubes themselves; and this in two ways—(1) from extreme pressure of congestion; (2) as an inflammatory exudation. In so far as increased pressure, whether from general conditions of the circulation (exercise, full meals), or from the pressure of leucocytal infiltration in the substance of the kidney, leads to distension of the glomerulus, this cause of albuminuria falls within the scope of the more generally accepted theory. But it seems possible that some transudation may take place, for instance, from the vasa recta into adjacent tubules. Similarly, if in an acute inflammation the substance of the kidney is infiltrated with inflammatory serum, it is held by some (Greenfield) that this escape into the tubules may contribute albumin to the urine.

The Presence of other Proteids in the Urine.—Besides serum-albumin, other proteids may be found in the urine.

Serum-globulin (or *paraglobulin*) commonly accompanies albumin and is precipitated by heat, by cold nitric acid, and by picric acid. It is often in excess in the urine of lardaceous disease, and may be detected by pouring the urine into a large bulk of distilled water, when the globulin is precipitated, or by saturating the neutralised urine with magnesium sulphate.

Nucleo-protein. Mucin.—If a few drops of acetic acid be added to the cold urine, especially if diluted, a precipitate may form, which was once thought to be mucin, and more recently a nucleo-protein derived from the renal cells. It is now stated (Mörner) that such precipitates are compounds of serum-albumin with chondroitin-sulphuric acid, with nucleic acid, and in cases of jaundice with taurocholic acid. If it occurs in nephritis or cyclic albuminuria, it is a mixture of euglobulin and fibrinogen (Oswald). According to these later views it is derived from the blood, and has the same significance as serum-albumin.

Albumoses also occur in the urine, generally in association with albumin, but sometimes without it. They are due mostly to absorption from purulent effusions, such as empyema, and large abscesses; to diseases of the liver, especially acute yellow atrophy; to ulceration of the bowel in typhoid fever or otherwise; to acute infectious diseases and acute inflammation, especially pneumonia; to scurvy, and to toxic and puerperal conditions. They may occur in large amount without albumin in some cases of chronic Bright's disease (granular kidney). Albumoses are precipitated by salicyl-sulphonic acid, re-dissolved on boiling, and again thrown down on cooling. Nitric acid, added gradually, gives a precipitate, soluble on heating, and reappearing in the cold. The albumose of Bence Jones, which occurs in some diseases of the bone-marrow (*myelopathic albumosuria*), is coagulated by a lower temperature than albumin, that is 58° C. (137° F.), as compared with 75° (167° F.), and the coagulum is re-dissolved as the temperature is raised to boiling. Strong hydrochloric acid throws down an abundant precipitate, which is only dissolved in very great excess of acid, but is soluble on boiling. In smaller quantity, an amount of albumose equal to ·5 per 1000 will give a white ring at the junction of the fluids, when the urine is floated on the acid (Bradshaw's test).

Peptones rarely, if ever, occur in the urine. They give the biuret reaction—a pink or rose colour when the urine is floated in a test-tube over a small quantity of Fehling's solution—but they are not precipitated by nitric acid, nor by saturation of the fluid with ammonium sulphate, which precipitates the other proteids. This and other salts may be used for the separation of the proteids, since ammonium sulphate precipitates all proteids, except peptones, sodium magnesium sulphate precipitates serum-albumin, and magnesium sulphate throws down serum-globulin.

HÆMATURIA

In acute nephritis the urine often contains blood or some of its constituents other than albumin. This is rarely in such quantity as occurs from calculus and growths in the kidney; but it sometimes gives the urine a bright-red colour, and more often a dirty-brown colour and turbid appearance from the presence of methæmoglobin or hæmatin. Generally with this there is a granular reddish-brown sediment. The presence of blood is determined with certainty in several ways.

The Microscope.—By this we can recognise blood discs, in cases where there is no suspicion, from the colour of the urine, that blood is present; if the urine is distinctly red, or brown and turbid, the discs will be there in abundance. From suspension in the urine they may have lost their biconcave form, and are often shrivelled, and have crenate edges, or may present protrusions of their substance. They remain visible for the longest time in acid and dense urines, but may be quickly dissolved in ammoniacal urine, or in urine of low specific gravity.

The Spectroscope.—The spectrum of urine containing oxyhæmoglobin shows two absorption bands in the yellow and green portions between Frauenhofer's lines D and E, the narrower, darker, better-defined band being nearer to D. Methæmoglobin gives three absorption bands, two in very nearly the same position as those of oxyhæmoglobin, and a third about half-way between C and D. Acid hæmatin shows a fourth band between E and F.

Heller's Test.—This consists in adding caustic potash or soda to the urine and then boiling. The phosphates which are precipitated are coloured pink or red by the hæmoglobin which they carry down with them.

The Guaiacum Test.—To the urine, in a test-tube, are added a few drops of tincture of guaiacum, and then about half a drachm of ozonic ether. Quickly or slowly, according to the quantity of blood, a blue colour forms at the junction of the fluids, and diffuses itself through the ether which floats on the surface; its appearance may be hastened by shaking the mixture. The test is not absolutely trustworthy. The urine of patients taking potassium iodide will give a blue colour, but it appears only slowly unless the iodide is in very large amount.

It is often desired to know in what part of the urinary passages the hæmorrhage has arisen. In renal hæmorrhage the blood is often intimately mixed with the urine; in bleeding from the bladder it is more separate; in urethral hæmorrhage the blood comes apart from the urine. The diagnosis is also helped by any coagula that may form: in renal hæmorrhage blood-casts of the renal tubules are found, in hæmorrhage from the ureter there may be long stringy coagula, and in vesical hæmorrhage large flat oval clots 1½ inches in length and 1 inch broad with fringed edges.

CASTS

These are solid bodies, which are detected by the microscope in the urine, and if sufficiently numerous form a sediment visible to the naked eye. Where they are too few to be easily detected, they may be found after centrifugalising the urine. They are cylindrical in shape, and from ·01 to ·05 mm. in breadth; but they vary still more in their length, which may reach 1 or even 2 mm., so as to stretch right across the field of the microscope, but is more often from five to ten times the breadth. Their connection with the kidney is proved by finding them after death, *in situ*, in the renal tubules. The following varieties are distinguished.

Hyaline Casts.—These are transparent colourless cylinders, with refractive properties so like those of the fluid in which they lie, that they are discovered with great difficulty, unless they are stained by the addition of carmine or iodine, or one of the aniline dyes, such as gentian-violet. They are homogeneous, soft, and flexible, straight or curved, and varying in length. Occasionally they have other deposits adherent to, or embedded in them, such as red blood-corpuscles, leucocytes, epithelial cells, granular masses, fatty granular globules, crystals of urates or oxalates, or particles of hæmatoidin.

Some of the varieties of casts described below probably have the same hyaline material as a basis, which is then completely covered by, or mixed up with, the other elements.

Hyaline casts are often spoken of as "fibrinous," but it appears that they are neither pure fibrin nor pure albumin, but can at present only be described as albuminoid. Most probably they are produced by the coagulation of a fluid exuded by the glomeruli, as they form with great rapidity—sometimes in a few hours. But they are thought by some to be due to an altered condition of the renal epithelial cells, or of leucocytes.

They occur in the different forms of nephritis, and in the congestion produced by heart disease; they are nearly always associated with the albuminuria of renal origin, but may precede for a few hours or days the appearance of the albumin, and may continue for a time after its disappearance.

Granular Casts.—These are not so transparent as hyaline casts, being sometimes like ground glass, sometimes darker and much more opaque.

Epithelial Casts.—These consist of cells of the renal epithelium held together by, or embedded in, the coagulable material which makes up the hyaline casts. The epithelial cells may be more or less distinct; and some have thought that they are more often swollen leucocytes than actually the cells of the renal tubules.

Fatty Casts.—These are either hyaline casts in which globules and granules of fat are embedded, probably from the disintegration

of epithelium in a state of fatty degeneration; or they actually consist of such fatty epithelial cells themselves.

Blood-Casts.—These result from the coagulation of blood that has been effused into the renal tubules; they are easily recognised by their colour, and by the size and close aggregation of the blood-corpuscles composing them. Their presence in a case of hæmaturia proves that the blood comes from the kidney itself.

Waxy Casts.—These are large and highly refractive casts, which are sometimes found in cases of lardaceous disease, but also in other chronic forms of Bright's disease. They often do not give the reactions of lardaceous material, and their nature is doubtful.

Though the character of the casts found in the urine must be determined to some extent by the condition of the kidney, conclusions on this last point must not be too hastily drawn from them. Frequently two or more forms are found in the same urine, and we must be guided by the predominance of one or the other. Hyaline and granular casts are found in all forms of nephritis, acute or chronic. Blood-casts and epithelial casts are most common in acute cases; fatty casts are most frequent in cases of chronic nephritis undergoing fatty degeneration.

Cylindroids.—These are large hyaline bodies with tapering or branching ends, a wavy outline, and longitudinal striation: they are probably threads of mucus, the undissolved residue of nucleo-proteid, and not really casts. They have no significance.

DROPSY

Two forms of dropsy may be recognised in renal disease.

One of these is identical with the dropsy which results from heart disease, and is, indeed, due to the secondary disturbances of the heart which subacute or chronic Bright's disease induces. In this, the dropsy occupies especially the lower extremities and the lower half of the body, while the face, arms, and upper half of the body may be free.

The other form is the characteristic *renal dropsy*, which is seen typically in acute Bright's disease, and involves the whole surface of the body and the great serous cavities. Frequently the first change noticed is some puffiness of the eyelids when the patient rises in the morning. This may subside in the course of the day, but if the feet be examined at night there is œdema just below the ankles. There is, indeed, a small quantity of effusion into the subcutaneous tissue, which always seeks, by force of gravity, the most dependent part. During the daytime it reaches the feet; in the recumbent position of sleep it diffuses itself generally, but is most pronounced in the loose tissues of the eyelids. If the patient takes to his bed, it will leave both the face and the feet, and accumulate in the tissue over the sacrum, which has now become the most dependent part. In more advanced cases the dropsy

becomes general, and the skin of the whole body is œdematous. The face is full and rounded, the eyelids are distended, and almost close the eyes; the limbs become enlarged, shapeless, and remind one of bolsters; the trunk is enlarged; the loose skin of the penis and scrotum is so stretched that the prepuce looks like a bladder, and the scrotum may attain the size of a fœtal head. Wherever slight pressure is applied, as by the finger of the doctor, or by the bands, strings, or folds of clothing, it produces by the displacement of the fluid a deep impression, which is only slowly effaced by the return of the fluid. This is called *pitting on pressure.* Even then the influence of gravitation on the distribution of the dropsy may be seen, for if the patient lies for any length of time on one side, the arm of that side will become more swollen than the arm which is uppermost; and the same will happen to the side of the face. When this general dropsy, or *anasarca,* is present, there is, as a rule, some effusion into the peritoneal cavity (*ascites*), into one, or more frequently both sides of the chest (*hydrothorax,* dropsy of the pleura), and it may be into the pericardial sac. Of these, the first is perhaps most often recognised; while the pericardial and pleural effusions may be comparatively slight.

If incisions be made into the skin, or if one or more Southey's drainage-tubes be inserted, a quantity of fluid will drain away, which may amount to eight or ten pints in a few hours, and the dropsical limbs will rapidly get smaller. The fluid is colourless, of low specific gravity (1007–1012), and contains a small quantity of albumin, inorganic salts, and urea. A feature which is constantly present in renal dropsy is a high degree of pallor—the lips are almost colourless, the cheeks are pallid, and the whole body has a waxy whiteness. This is partly due to the dilution of the blood and its poverty in red corpuscles; partly to the distension of the skin and subcutaneous tissues.

The typical anasarca of renal disease is by no means so easily explained as the dropsy of heart disease. In this latter it is clear that overfilled vessels on the venous side of the heart are unable to take up the fluid, which naturally transudes into the subcutaneous tissue. In renal dropsy an analogous mechanical explanation is held by many. As a rule, much dropsy coincides with decided decrease in the quantity of urine secreted, and it is an obvious suggestion that the liquid which cannot pass out by the kidneys escapes into the subcutaneous tissue; and in cases that recover, it is common to observe that the dropsy diminishes coincidently with an increase of urine. It might then be supposed that the retention of water by the kidneys, while the patient drinks the same as in health, increases the arterial tension throughout the body, and this relieves itself by transudation into the tissues. But suppression of urine undoubtedly occurs in some other ways without producing anasarca, and in one form of dropsy urine is abundant.

Other views have been expressed with regard to renal dropsy:

One, that the exudation is really of an inflammatory nature; another, that the specific gravity of the blood is lowered in renal disease, and hence transudation is facilitated—but the experimental production of hydræmia does not lead to dropsy. Many think that the fault lies in an abnormal permeability of the capillaries, damaged by the condition of the blood; and recently dropsy has been attributed to the abnormal retention of chlorides, which need the presence of more water or lymph for their dilution to a physiological strength.

In chronic renal disease (granular kidney) the urine is at first abundant; and dropsy is slight or none until the urine becomes scanty from other renal changes, or until the heart becomes secondarily involved, and a cardiac dropsy results.

CARDIO-VASCULAR CHANGES

These are, in acute cases—(1) High arterial tension, and (2) dilatation of the left ventricle; in chronic cases—(1) High arterial tension; (2) thickening and atheromatous change in the arteries; (3) hypertrophy and dilatation of the heart.

Arterial Tension.—In acute nephritis, quite early in the history of the case, the pulse will be found abnormally hard, showing a pressure of six or eight ounces or more on the sphygmograph, or from 170 mm. to 250 mm. on the sphygmomanometer. The pulse tracing is often highly characteristic, the tidal and the dicrotic wave being both very pronounced, and so forming an outline like a hill with three nearly equal peaks (*see* Fig. 51). Coincidently with this the cardiac sounds are modified; the first is reduplicated or broken, and the second is accentuated, or ringing, over the base of the aorta. In some severe cases, after a certain duration, dilatation may be observed, the impulse being under the nipple or even outside it; and the heart may become irregular in its action. If the illness is much prolonged, there is a tendency to the hypertrophy which is seen in chronic cases.

High arterial tension is also the rule in chronic Bright's disease; the pulse is hard and resists compression, and yields a tracing in which the tidal wave is high and long, giving a flat top to the outline, but in which the dicrotic wave is only slightly marked (*see* Fig. 51).

Changes in the Vessels.—Atheromatous changes take place in the artery walls, and are no doubt in part the result of the strain to which the vessels are put; but they are neither constant in, nor peculiar to Bright's disease. Much more characteristic is the more or less uniform thickening (arterio-sclerosis) which affects the small arteries all over the body, as well as the vessels in the kidney itself; and consists chiefly of a hypertrophy of the middle or muscular coat.

Changes in the Heart.—Hypertrophy of the heart is a very frequent change in chronic Bright's disease, and in granular kidney it reaches its greatest extent. It may show itself by the usual

physical signs—namely, displacement, and heaving character of the impulse. The weight of the heart varies from seventeen to twenty-eight ounces, and the hypertrophy in the more marked cases affects the right as well as the left ventricle. In advanced cases it is accompanied by dilatation, and this has an important bearing upon the clinical features of the illness. With its occurrence, the circulation, already obstructed by the high arterial tension, fails more and more, the action of the ventricle becomes irregular, regurgitation takes place through a leaking mitral orifice, and eventually the complete picture of a primary valvular disease may be developed, with a mitral murmur, engorgement of the lungs and liver, ascites, and dropsy of the lower half of the body.

The cause of the cardiac hypertrophy in renal disease has been no less debated than the many other conditions in this interesting disorder. Bright believed that the heart hypertrophied from the irritation of its cavities by the impure blood. Another view, long and widely entertained, has been that the blood, poisoned by urinary constituents, circulated with difficulty through the capillaries of the body, and the left ventricle necessarily hypertrophied to overcome the resistance thus created. Johnson supposed that the muscular coat of the arteries hypertrophied, in order to prevent the poisoned blood reaching the tissues; and that the heart hypertrophied in consequence of this arterial resistance. Others (Traube, Cohnheim, Fagge) thought that the obstruction lay in the kidney, in consequence of a diminution either of its vascular area or of the substance available for excretion: the thickening of the systemic arteries was then regarded as an attempt on their part to protect the tissues from undue pressure. By some, chronic Bright's disease, in the form of granular kidney, is regarded as a general and simultaneous affection of the heart, the arteries, and kidneys; but if this be true, we still have to account for the precisely similar changes which occur in chronic parenchymatous nephritis, in which case the renal disorder undoubtedly precedes the other symptoms.

OCULAR CHANGES

In chronic forms of Bright's disease, especially in granular kidney, and also in lardaceous disease, changes of much importance take place in the fundus of the eye. They consist of—(1) œdema of the retina, causing opacity and swelling; (2) white spots or patches in the retina; (3) hæmorrhage into the retina; (4) papillitis; and lastly (5) atrophy of the retina and papilla, the result of the preceding inflammation. These are generally grouped under the term *albuminuric retinitis*. In addition there may be (6) changes in the renal arteries.

The most frequent and characteristic are the white spots, which are often of a glistening or silvery white colour, at first very small, though subsequently enlarging, partly by coalescence. They are most numerous in the neighbourhood of the optic disc, and

especially around the yellow spot, where they may have a more or less radiating arrangement. Often a large number are grouped together so closely as to resemble a piece of mosaic. They are due to a degenerative and fatty change in the elements of the retina, especially in the nerve-fibres, but also in the corpuscles and Müller's fibres. The nerve-fibres become thickened and swollen, and varicosities form, which are filled with fat globules; compound granule-cells are also found in their deeper layers.

The hæmorrhages vary in size, are often small, sometimes elongated, pointed and divided, or "flame-shaped," radiating from the optic disc as a centre. They are mostly seen in conjunction with the white spots.

Neuritis or papillitis is shown by the swelling of the disc, the opacity or blurring of its edge, the partial or complete concealment of the retinal vessels, especially the arteries, and the filling and tortuosity of the veins. The condition varies in different cases. Sometimes it is so great as to resemble the neuritis of marked cerebral disease. More often there is but slight prominence, but diffused opacity spreads far on to the surrounding retina. In many cases the papillitis, spots of degeneration, and hæmorrhages co-exist, sometimes one, sometimes the other, being more pronounced.

The changes in the retinal arteries may co-exist with renal retinitis, or even precede it, and are a part no doubt of the general arterio-sclerosis. The arteries are narrower than usual, varying in breadth and with fusiform enlargements: they frequently show a brilliant central streak of metallic lustre, and the streak may be broken, patchy, or in dots. These have been called *silver wire arteries*.

Probably, in the majority of cases, the retinal changes persist until the patient's death; but improvement may take place, neuritis subsiding, diffused blood being absorbed, and even the spots of degeneration disappearing. Loss of vision is more or less in proportion to the extent of the changes, and of the implication of the yellow spot. In the earlier stages there may be no appreciable loss, and total blindness is quite rare. On the other hand, a temporary absolute blindness may occur as a part of uræmia without any retinal changes.

HÆMORRHAGES

Partly from the change in the vessel walls, partly from the hypertrophy of the heart and the high arterial tension, hæmorrhages are frequent in Bright's disease. Retinal hæmorrhages have just been described, epistaxis is common, and purpura and bleeding from the stomach and bowel may occur. The most important is cerebral hæmorrhage, which is a frequent cause of death in chronic renal disease.

SECONDARY INFLAMMATIONS, AND OTHER LESIONS

Both in acute and chronic nephritis there is a tendency to inflammation of the serous membranes. Pleurisy is the most common; pericarditis is often the precursor of a fatal termination; acute peritonitis is perhaps more rare, unless it follows tapping the abdomen. Chronic inflammation of the peritoneum covering the liver (perihepatitis) has already been referred to in connection with Bright's disease. Bronchitis is a common, and endocarditis an occasional, complication; pneumonia not infrequently occurs towards the end. Several lesions of the skin may complicate Bright's disease—namely, (1) pruritus and urticaria, the former especially in early stages; (2) eczema; (3) an acute general dermatitis, with free desquamation, not unlike exfoliative dermatitis; (4) erythema læve, a condition of redness in more or less continuous patches, affecting the dropsical limbs; (5) erysipelas; (6) purpura or hæmorrhage, followed by necrosis and ulceration, conditions which have also been observed in the alimentary mucous membranes; and (7) the rare excretion of urea with the sweat (uridrosis) in sufficient amount to form crystals (probably not entirely of urea) which give the skin the appearance of having been dusted with flour or pounded sugar.

URÆMIA

This term is applied to a number of nervous symptoms which arise in different forms of Bright's disease.

Acute Symptoms.—The most striking of these are *uræmic convulsions* or *uræmic eclampsia*. These have a very close resemblance to, indeed may be identical with, the ordinary attack of epilepsy. There is often a short tonic stage, and then general clonic convulsions of all the muscles of the limbs, face, eyes, and trunk. The face becomes livid, there is frothing at the mouth, the saliva may be tinged with blood, and the pupils are dilated. After some minutes the convulsions subside, and the patient lapses into a state of *coma*, from which he may again pass into convulsions; and these are repeated again and again with intervals of complete coma. During the convulsion the respiration is hurried, and the pulse is small and quick; the temperature is variable, and it may reach 104°, or higher. Thus, convulsion and coma succeed one another; but either may occur separately. Sometimes coma comes on quickly; or more slowly, drowsiness gradually increasing to stupor and complete unconsciousness in a few hours. A temporary *paralysis* is sometimes observed.

Delirium or mania is another form of disturbance which results from uræmia, but it is not so common as the convulsive and comatose symptoms; it may follow the fits.

It is also after convulsions that the blindness (*uræmic amaurosis*), to which I have already referred, generally occurs; it rarely pre-

cedes the fits, or happens without them. It may last from one to three days, and frequently passes off entirely. As the pupil reactions are unaffected, it is probably due to the influence of toxins upon the higher visual centres.

Deafness may be also noticed.

Chronic Symptoms.—These are headache, twitching of the muscles without loss of consciousness, recurrent attacks of *dyspnœa*, anxiety, and restlessness, or somnolence and stupor, itching of the skin, *vomiting* and *diarrhœa*. Vomiting may be referred to irritation of the nerve-centres, though it has been thought by some to be due to the presence of urea or carbonate of ammonium in the gastric secretion. The contents, however, of the stomach are usually acid. Diarrhœa, though often accompanying vomiting, is sometimes associated with decided lesions in the intestinal mucous membrane, which may be œdematous or ecchymosed, or in a condition resembling dysentery. The symptom can then scarcely be classed with the other uræmic conditions.

The dyspnœa sometimes resembles spasmodic asthma, and comes on at night. Sometimes stridulous breathing occurs exactly resembling that of laryngeal obstruction or tracheal stenosis, the patient being conscious at the time. Cheyne-Stokes breathing is a very common event in chronic uræmia, often for many weeks preceding death. A group of symptoms different from those above described occurs in obstructive suppression of urine (*see* p. 960), and is sometimes called latent uræmia.

Results.—Though uræmic symptoms indicate a very grave condition, and generally coincide with a considerable diminution in the secretion of urine, and especially of urea, still they may disappear completely. This is especially the case in acute nephritis, where the condition of the kidney may be only temporary; whereas the less acute symptoms of uræmia in chronic nephritis not infrequently persist to the fatal termination. Here, also, an attack of coma often ends the scene; but it is generally a succession of fits rather than a single one that is fatal, whether in acute or chronic disease.

Pathology.—The most plausible explanation of uræmia is that it is due to the retention in the blood and tissues of some of the excrementitious matters that ought either themselves, or in some changed form, to be excreted by the kidneys. Analyses of the blood in uræmia have not generally shown much urea; but urea is found in the gastric and intestinal secretions, in the dropsical fluid under the skin, and rarely in the sweat. But on various grounds it is clear that neither urea, nor ammonia, nor any single constituent of the urine is the sole cause of uræmia. Bouchard regards it as the toxic effect of several urinary constituents, not necessarily the same in every case of uræmia. Others attribute it to the accumulation of toxic products of intestinal decomposition, which normally should be excreted by the kidneys.

Bradford has shown that the experimental removal of large

portions of the kidney substance caused the death of the animal with its muscles loaded with urea and other nitrogenous extractives, although no retention of urea or water occurred. Hence there must have been increased tissue degeneration, which it may be conceived occurs also when the kidneys are diseased beyond a certain point.

INADEQUACY OF THE RENAL FUNCTIONS

In the course of various forms of Bright's disease and nephritis, it becomes important to estimate the power of the kidney as a secreting organ, since the amount of albumin passed per diem is not by any means an accurate measure of the damage suffered by the organ. The following are some of the methods by which an estimate is attempted.

Measurement of the Nitrogen in the Urine.—The solids contained in the urine, which can be ascertained as shown above (*see* p. 889), give but a rough idea of the nitrogen-excretion, since one-half of them are salines, and the relative proportion of the urea and salines varies from day to day considerably. The urea, which contains 85 per cent. of the urinary nitrogen, and of which the normal daily average is about 500 grains, can only be rightly estimated by analysing a specimen from the mixed urine for the whole day; and this process must be repeated several days successively to eliminate the effect of variations. The urea-excretion is diminished in almost any form of illness, except diabetes, on account of the diminished intake of food, and no charge can be made against the kidney unless the daily amount falls to about 250 grains in a man, or 200 grains in a woman, on ordinary diet.

Methylene Blue.—If 1 cubic centimetre of a 5 per cent. solution of methylene blue is injected subcutaneously into a healthy person, the bladder being at the same time emptied, and the urine is voided every half hour, or hour, afterwards, the first appearance of a blue colour will be within one hour, the colour will be at a maximum in three or four hours, and will have disappeared in twenty-four or thirty-six hours. Defective excretion by the kidney is shown by absence of colour at the end of an hour, by delay of the maximum colour beyond four hours, and by persistence of the colour for five or six days.

Phloridzin.—The injection of phloridzin under the skin or into muscle causes the excretion of sugar by the urine: after the injection of 1 cubic centimetre of a ·5 per cent. solution sugar will appear in from half an hour to an hour, and will disappear in from two to four hours. Delay beyond these times indicates defective action of the kidney.

ACUTE NEPHRITIS

Ætiology.—The several toxic and microbic causes of nephritis have already been mentioned. But it must be allowed that a large number of cases of acute nephritis arise, like pneumonia and pleurisy, apparently from cold or spontaneously; and where as in those diseases the pneumococcus or the tubercle-bacillus seems to be the efficient cause, the microbic element in nephritis is still very obscure, and the exact relation between it and cold has yet to be revealed. The influence of cold seems to be greater when associated with damp: and the risk is increased if the individual is much exhausted from exertion or other cause, if he is asleep, or if he is under the influence of alcohol. Adults are much more liable than children to have nephritis from this cause.

The poison of scarlatina is another extremely common cause of acute nephritis, and in this case children are more often the subjects than adults. As a rule, the symptoms are first observed during convalescence, and here again they have been attributed to cold; but nephritis also arises in cases which have been most carefully nursed, and have not been exposed. Sometimes it commences before convalescence is established.

Several other febrile diseases are from time to time, but much less frequently than scarlatina, the causes of acute nephritis. Of these cases also a very small proportion develop into a typical Bright's disease, with dropsy, secondary inflammations, uræmia, &c.; but the majority present only temporary changes in the urine, with but slight constitutional disturbance. They are measles, variola, cholera, varicella, typhus, enteric fever, pneumonia, relapsing fever, and erysipelas. Diphtheria is frequently accompanied by albuminuria, sometimes clearly indicative of nephritis. With these may be classed tubercle, syphilis, and septic conditions from purulent absorption.

Pregnancy is not infrequently the cause of a nephritis, which may be of the most severe type, with uræmia and retinal changes.

A number of substances taken internally set up irritation of the kidney, which, according to the dose, may be severe congestion, or may amount to a definite nephritis. They are cantharides, turpentine, potassium nitrate, salicylic and carbolic acids. Indulgence in alcohol, though probably more often contributing to the chronic form of nephritis, occasionally seems to produce an acute inflammation. Uranium nitrate has been used experimentally to produce nephritis in animals.

Nephritis may also occur by infection from various lesions of the lower urinary passages (*consecutive nephritis*), or from organisms present in the urine (*bacilluria*).

Symptoms.—These are in some cases almost confined to an alteration in the urine, of which the main feature is the presence of albumin; often, also, the urine is scanty, micturition is frequent,

and blood and some casts may be present. With this there may be some rise of temperature, a feeling of malaise, and perhaps pain in the loins. In this latter case there can be no doubt of the existence of a nephritis, but if the urine is alone affected, it is often attributed to severe *congestion;* thus, in the course of acute febrile illnesses a mild albuminuria occurs, which is probably due either to congestion or to a slight degenerative change in the renal structures.

But in the cases commonly included under the name of acute Bright's disease, the first event is dropsy, which occurs in the manner described in the account of that symptom (*see* p. 907). Sometimes it appears within a few hours after exposure to cold, or it is first noticed in the stage of convalescence from scarlet fever. Almost simultaneously the urine becomes affected, and there may be a rigor and some discomfort or pain in the loins. The urine is scanty, the quantity diminishing to ten, eight, or six ounces daily, or even less; it is acid and irritating, so that it is frequently voided in quite small quantity; its specific gravity is high, from 1025 to 1030; it is turbid, and it has a colour which is due to the presence of fresh or altered blood, and is dusky brown, deep brown, "porter-coloured," pink, or distinctly red, according to its quantity and condition. It deposits a sediment consisting of fresh or altered blood-corpuscles, or fragments of them, renal epithelial cells, hyaline, granular, epithelial, or blood casts, granular *débris*, and it may be after some time uric acid crystals. Albumin is always present, and generally in large quantities, forming a thick curdy deposit on boiling; or the urine may become actually solid on heating, so that the test-tube may be inverted without a drop running out. The amount of albumin in any specimen varies from ·5 to 1 per cent., and the quantity discharged daily ranges from 100 to 400 grains. It must be understood that this is, in most cases, far in excess of what would correspond to the small amount of blood contained in the urine. The urea is remarkably diminished, it may fall to half its normal quantity or less daily, and the phosphates and chlorides are also reduced.

Quite early the pulse becomes hard and tense, and the heart-sounds are modified as described. In severe cases the heart dilates, and the impulse may be displaced outwards.

In many instances this constitutes the whole of the illness. After a few days, or a week or two, improvement sets in, the dropsy begins to diminish, the blood disappears first from the urine, then after some longer time the albumin; the specific gravity diminishes, the quantity is increased, reaching, it may be, sixty, seventy, or eighty ounces daily, and eventually, with the exception of some pallor and weakness, the patient returns to his normal condition.

But the course of acute nephritis may be less favourable in several ways. In some cases quite early the urine is almost entirely suppressed. For several hours, or a day, not a drop is passed, or only a few drachms of dark brown, opaque, highly albuminous urine. In these circumstances uræmic convulsions are very apt to

occur, which may be fatal, or subside coincidently with an increase in the secretion of the urine.

In other cases dropsy becomes acute, and effusion takes place into the peritoneum, pleural cavities, and pericardium. The lungs at the same time are partly œdematous, partly compressed by the pleural fluid, and death may take place from interference with their functions. Sometimes the excessive distension of the skin, especially in the lower extremities, causes gangrene and sloughing, or erysipelas occurs around the punctures which have been made to relieve tension; and death in either case may follow.

In other instances, again, the acute serous inflammations, pleurisy, pericarditis, and peritonitis, are fatal; or acute œdema of the glottis brings about asphyxia, unless promptly relieved by tracheotomy.

Lastly, the condition may remain for a long time unchanged, and eventually it becomes subchronic or chronic, and must be classed with the form of nephritis to be described presently.

Morbid Anatomy.—In some cases, especially those in which the glomeruli are mainly affected, the kidney may be but little larger than normal; the cortex is dark, and the glomeruli are pale. In other cases the kidney is more or less swollen, it may be twice its normal size, it has a rounded form, is tense and elastic, the capsule strips readily, and the surface is paler than normal. On section, the cortex is considerably swollen, and of grayish-red colour, while the pyramids are dark red from congestion. Here and there are bright red spots: some of them are congested glomeruli, others are small hæmorrhages. In yet other cases the kidneys are intensely congested, dark red or chocolate in colour, dripping with blood, and showing on section a still more extreme congestion of the pyramids.

Microscopically, one can distinguish changes which may be simultaneous in the tubules, in the interstitial tissue, and in the glomeruli; and if one speaks of a glomerular, of an acute interstitial, or of a tubal nephritis, it must be understood that, though one of these structures is especially concerned, the others are probably also affected, though in a much less degree.

In the tubules the epithelial cells become swollen and granular, and in more advanced conditions are filled with fat granules, or become necrosed, and are separated from the wall of the tubule (desquamation). The tubule is then filled or even distended by accumulations of such altered cells, mixed with albuminous fluid, leucocytes, and granular *débris*, and here and there blood escapes into them from distended intertubular vessels. Many tubes, both in the cortex and in the pyramids (*e.g.*, Henle's loops), are filled with hyaline or other casts. The distension of the tubes gives a pale colour to the portion of the kidney affected, partly from the opacity of the fat granules, partly from compression of the intertubal vessels; on the other hand, such compression increases the congestion of the glomerular tufts.

The size of the largest kidneys is perhaps due to interstitial

exudation. This may be either a general infiltration with inflammatory fluid, or the extravasation, more or less irregularly, of leucocytes from the blood-vessels. They are most abundant around the Malpighian capsules, and the intertubal vessels. The glomerular changes (*glomerular nephritis*) consist of the distension of the Malpighian capsule with a fine granular mass in which are embedded numerous small angular nuclei. Occasionally, hæmorrhage takes place. The glomerular tuft is compressed by the effusion, and the circulation and secretion of water are materially interfered with. A hyaline transformation of the walls of the afferent arteries has also been described as a constant change in the glomerular nephritis of scarlatina.

Diagnosis.—This rarely presents any difficulty. The sudden appearance of a general dropsy, with scanty, albuminous urine, in one previously quite healthy, or recovering from scarlatina or other fever, can scarcely be mistaken for anything else. In the absence of dropsy, the mere presence of albumin in the urine is not sufficient for the diagnosis. Turbid brown urine, abundance of albumin and casts, and constitutional disturbance will then show that there is nephritis, but for this the name of Bright's disease is not generally employed. If there is a small quantity of albumin only in the course of an acute disease, this may be due to a change in the epithelium, not usually regarded as constituting nephritis, though it is not essentially different from what actually takes place in the typical conditions. Lesions of the *lower urinary passages* may cause bloody urine and albuminuria. Dropsy and casts are absent. *Hæmoglobinuria* causes a red urine, in which, however, there are no blood-corpuscles. Rarely the dropsy of nephritis occurs without albuminuria, especially in children. Such cases must be distinguished from some insidious diseases (tuberculosis, leuchæmia), which have other characteristic lesions.

Prognosis.—It is, on the whole, favourable, as there are a great number of mild cases which recover in a few weeks, and some even after months. Some cases are fatal in the acute stages, and others run on into chronic nephritis. The unfavourable indications are excessive dropsy, very scanty or suppressed urine, very high tension of the pulse, or the later condition of feeble pulse with obviously failing heart, hydrothorax, serous inflammations, and uræmia. But there is scarcely any of these serious dangers from which recovery may not take place, and sometimes, after living for months on the verge of death, the patient may ultimately get quite well.

Treatment.—The direct treatment of the inflamed kidneys, either locally or by drugs, is, as a rule, not attempted. Only exceptionally, where there is much lumbar pain or free hæmorrhage from the kidneys, may dry-cupping be employed to the loins, or for the former symptoms a hot poultice or fomentations.

The usual treatment of nephritis consists in keeping the patient at rest, removing all sources of irritation, limiting the supply of food likely to put a strain upon the excreting functions of the kidney, and

supplementing these functions as far as possible by the use of other secreting organs—the skin and the bowels. In addition, we have to deal with special symptoms and complications as they arise. Rest in bed in a warm room diminishes the strain upon the heart and the need for highly albuminous food, while the secretion from the skin is likely to be promoted.

The diet is best confined to milk alone, or milk and soda-water. All kinds of meat, fish, and eggs should be rigorously excluded, and only in milder forms or after the acute stage has passed may a moderate amount of farinaceous food—gruel, arrowroot, and, later, toast, and a little bread and butter—be allowed, and with progressing convalescence a gradual return to meat diet, after beef-tea, broths, eggs, and fish.

In mild cases the medicinal treatment should be a saline diaphoretic, such as liquor ammonii acetatis or liquor ammonii citratis, in doses of 3 or 4 drachms every four hours; and the bowels may be kept active by occasional purges of pulvis jalapæ co., 20 to 40 grains, or senna and sulphate of magnesia. Lemonade may be given as a beverage, or the Imperial drink, which may act mildly on both the bowels and the skin. This consists of a drachm or a drachm and a half of cream of tartar dissolved in a pint of boiling water, flavoured with lemon peel and sugar, and drunk, *ad libitum*, when cold.

A diminution of the dropsy is sometimes obtained by the use of a diet as free as possible from sodium chloride, for one cause of dropsy is the retention of water in the tissues by the sodium chloride which the diseased kidneys fail to eliminate. But if pushed too far this method may cause much prostration. If dropsy is considerable, or if there are fears, either from the scantiness of the urine, or from headache, drowsiness, and twitching, that uræmic convulsions are imminent, the treatment by purgation and diaphoresis must be more decided. Diaphoresis should be promoted by the vapour bath, by the hot-air bath, by the use of jaborandi, or by pilocarpine. The *vapour bath* is administered by raising the bed-clothes from the patient by means of a low cradle, and fitting them close about his neck and round the sides and end of the bed. Into the space thus formed projects the long tube from a kettle of water, placed at the foot of the bed, and kept boiling by a spirit-lamp or gas flame. The steam thus brought into contact with the patient's body promotes free perspiration. Or a *hot-air bath* may be given by burning a spirit-lamp under a funnel connected with a tube similarly placed. The exposure should be from fifteen to twenty minutes; but it is desirable to take the temperature of the patient, as it may be inconveniently raised, if free sweating does not occur.

The most efficient diaphoretic is the nitrate of pilocarpine, of which $\frac{1}{6}$, $\frac{1}{4}$, or $\frac{1}{3}$ grain should be injected subcutaneously once a day.

In extreme dropsy, especially where the skin is tense and threatens to become inflamed or to slough, the dropsical fluid may be removed under antiseptic conditions, either by small incisions with a lancet

or punctures with the needle, or by the use of Southey's tubes. Two or more of these may be placed in each leg, and by this means several pints of serum may be withdrawn in a few hours. Only occasionally, in extreme cases, is it desirable to tap the abdomen or to aspirate the pleural cavity.

If uræmic convulsions set in, the patient's tongue should be protected as in epilepsy, by placing a piece of stick between the teeth, and pilocarpine nitrate should be at once injected under the skin. A few whiffs of chloroform will check the convulsions quickly, and though they may return on the removal of the anæsthetic, the effect may be kept up by its occasional use in small quantities, and thus a great deal of the violence of the fit is prevented. Bleeding from the arm will also sometimes stop them, and though it is, as a rule, undesirable that patients with pronounced renal disease should lose much blood, this remedy may be properly used if the convulsions are violent and persistent. The blindness which may occur after convulsions generally recovers of itself. Coma should be treated by purgatives and by pilocarpine. Vomiting will require effervescing mixtures, dilute hydrocyanic acid, or tincture of iodine in 2 or 3 minim doses every hour.

During convalescence iron should be given to restore the condition of the blood, but it is not well to use it in the early stages. The perchloride, and the ammonio-citrate, are suitable preparations. Attempts to lessen the amount of albumin by drugs, such as lithium citrate, sodium benzoate and tannate, strontian lactate and others have not been very successful. The patient throughout should be carefully kept from cold, and should, on getting up, wear warm clothing with flannel next the skin.

CHRONIC TUBAL NEPHRITIS

Ætiology.—This condition is by many described as exclusively a late stage of acute nephritis; but since it sometimes occurs spontaneously, and often insidiously, it seems better to treat of it independently. In cases which are chronic from the first the ætiology is by no means always clear. It has been attributed to cold and damp acting over long periods of time, as, for instance, from residence on damp soils; and a predisposition to its occurrence possibly arises from habits of intemperance, overwork, mental exertion, or sexual indulgence. Toxic and infectious agencies are very probable causes, though it is not generally evident what these are in particular cases; malaria, syphilis, and tuberculosis may be mentioned in this connection. Cases of this kind are more common in persons of middle age; rare in children and old people; and males are more liable than females.

Symptoms.—If the disease follows acute Bright's disease, there is a continuance of the symptoms already described: General dropsy, effusion into the serous cavities, scanty albuminous urine,

high tension of the pulse, and cardiac hypertrophy or dilatation. Or there may be an apparent recovery from the acute attack, and after a short interval the symptoms recur.

In primarily chronic cases the beginning is more or less insidious. Pallor, loss of appetite, nausea, headache, and frequent micturition are the first signs, and then œdema may be observed in the lower extremities at night, and around the eyelids in the morning. The dropsy gradually increases, and the case is then almost identical with the acute disease.

The urine is scanty, highly albuminous, sometimes bloody, is deficient in urea, and contains casts. Its quantity varies from ten to twenty-five ounces, though in later stages for a time it may be more abundant. The albumin forms a precipitate which occupies one-third to one-half of the urine boiled, really amounting to 2 or 3 per cent. by weight, or a daily discharge of from 100 to 400 grains. The specific gravity is at first rather high (1015 to 1025), but later, with the progress of contraction, it becomes much lower. The casts are granular, hyaline, and epithelial; and fatty casts are numerous in the cases of large white kidney with much fatty degeneration of the renal epithelium. They are accompanied by leucocytes, loose epithelial cells, and granular *débris*. In some cases the urine is frequently bloody (*chronic hæmorrhagic nephritis*). The amount of urea is always below the normal, and has the same importance with reference to the onset of uræmia as in the acuter forms of disease.

As the case progresses the heart becomes hypertrophied, and the vessels are thickened. The impulse of the heart is displaced outwards, but the œdema of the thorax may prevent its being felt; the aortic second sound is accentuated, the first sound is frequently reduplicated, and the pulse is one of high tension. The retinal changes already described are observed in a certain proportion of cases; while secondary inflammations, chronic uræmia, or uræmic convulsions and coma may at any time occur.

In the majority of instances, the end is fatal, and is brought about in the same way as in acute nephritis, by uræmia, pleurisy, pericarditis, pneumonia, œdema of the lungs, œdema of the glottis, increasing dropsy, or inflammation or sloughing of the integuments; and this is commonly in from six to eighteen months, though some cases go on for two or three years. If this does not occur, either the patient appears to recover completely, or the kidney becomes contracted, and the symptoms gradually acquire the characters of a chronic interstitial nephritis. Neither event is common.

Morbid Anatomy.—The *large white kidney*, or inflammatory fatty kidney of chronic tubal nephritis, is larger than normal—the two together may weigh as much as twenty-eight ounces, often they weigh fifteen or sixteen. It is smooth on the surface, and the capsule strips easily; it is of yellowish or grayish-white colour, and is covered with venules radiating from a central point (stellate veins). On section, the cortex is broader than normal, of the same colour

as the surface with an appearance of coarse striation, while the pyramids are more or less dark-red. Here and there are red spots, due to hæmorrhage into the tubes, and these are in some cases so abundant as to justify the term *hæmorrhagic nephritis*. Microscopically it is seen that the convoluted tubes are filled with epithelium in a state of fatty and granular degeneration, desquamated from the surface, as well as separate fatty granules; epithelial and granular casts occupy the straight tubes. This distension of the tubes with opaque material, and the compression they exert on the vessels, account for the white or gray colour of the cortex. The intertubal tissue is irregularly infiltrated with leucocytes, and the Malpighian capsules are thickened. Lastly, within the capsules, the glomerular tufts are compressed by exudation and the proliferation of the epithelium covering them.

Diagnosis.—The distinction between an acute nephritis and the first stage—large white kidney—of the chronic nephritis, which succeeds it, must be purely arbitrary; from four to six months may be regarded as the duration of the acute condition. In any case of less duration the diagnosis must depend on the history of sudden or insidious onset, and the previous occurrence of a cause, such as scarlatina, or its absence.

After a longer period, chronic tubal nephritis may be confounded with chronic interstitial nephritis, with lardaceous disease, or with the albuminuria of primary heart-disease (*cyanotic induration*). From a primary *chronic interstitial nephritis* it is distinguished by its history, the early appearance of dropsy, the age of the patient, which is mostly younger, the early scantiness of urine with abundant and often fatty casts; and from *lardaceous disease* by the absence of such causes as prolonged suppuration, phthisis, or syphilis, and by early scantiness of urine and abundant deposit. The similarity between this and heart-disease may be close, though not so much so as between heart-disease and chronic interstitial nephritis. This subject will be discussed more fully under the last head. Here it may be sufficient to state that the history, the general character of the dropsy, and the mostly large quantity of albumin will help to distinguish primary renal disease from secondary albuminuria.

Treatment.—This is not essentially different from that of acute nephritis. Rest in bed, flannel clothing to promote warmth and a free action of the skin, and milk diet, are actually essential in the early stages. The bowels must be kept active, and diaphoresis excited. The treatment for urgent symptoms is the same as for acute nephritis, except that venesection may have to be used with more caution in view of the marked anæmia. This last symptom in prolonged cases should be met by the use of iron preparations, such as the iodide, tartrate, or ammonio-citrate. In the more chronic forms, with albuminuria but little dropsy, benefit may be derived from residence in warmer climates—Bournemouth or Tenby in the British Isles, the South of France, Italy, or Egypt.

Cases of chronic Bright's disease have been treated by Edebohls

in America, by exposure of the kidney, decapsulation, and fixation of the organs: and the results of this surgical treatment have sometimes appeared to be good.

CHRONIC INTERSTITIAL NEPHRITIS

This is generally recognised as occurring in two or more varieties: the pale granular or contracted white kidney; the red granular or gouty kidney; and a form which is consecutive to obstruction of the urinary passages (*see* p. 928).

PALE GRANULAR KIDNEY

The possible origin of this in a condition of chronic, subacute or acute tubal nephritis has already been mentioned; but it is believed to arise much more often as an independent lesion without any preceding acute stage. The subjects are often persons in early adult life. The onset is insidious, dropsy is absent, albumin is abundant, and the amount of urine is often normal or even in excess: occasionally, hæmaturia occurs. The patients suffer from headaches, anæmia, and sickness; and have high-tension pulses and the cardiac phenomena associated with high arterial tension. Only late in the case may dropsy supervene, and uræmia is a frequent mode of termination.

Morbid Anatomy.—The size of the organ varies: it may be normal or not much smaller, or it may be considerably reduced. Its surface is rough or granular, the capsule slightly adherent, the colour yellowish white, or more or less mottled with red in the areas of depression between the granules. The cortex, on section, is narrower than normal, and is not so white as in the "large" kidney. Microscopically, the intertubal tissue is partly infiltrated with leucocytes, partly changed into connective tissue, which is more or less uniformly distributed; and it is by the contraction of this tissue that the granular condition is brought about, and the organ becomes smaller. Of the tubes some still contain altered epithelium and *débris*, others are atrophied from pressure; from others again small cysts have been formed, similar to those so common in the red granular kidney. Many glomeruli, also, are atrophied, or changed into fibrous nodules; and the small arteries are thickened. Lardaceous changes of slight extent are sometimes present.

The **Prognosis** is bad, and the duration of life probably much shorter than in cases of red granular kidney. Still one must recognise that some young patients with chronic albuminuria, who may reasonably be regarded as having pale granular kidneys, do occasionally get well after months or years.

Treatment.—This must be conducted on the same lines as that of the next form.

RED GRANULAR KIDNEY

This is the form of chronic interstitial nephritis to which the names *gouty kidney, primary contracted kidney, cirrhosis of the kidney, indurative nephritis, renal sclerosis, renal fibrosis,* and *arterio-sclerotic kidney* have been by different writers applied.

Ætiology.—It is a disease of middle and advanced life, occurring with extreme rarity under twenty years of age, and most frequently between forty and sixty; but it has been seen in infants, and even in the new-born. It affects males more than females. From its very slow development, it might be expected that the causes would be such as were in continuous action. The most important are gout, whether latent or developed, and chronic lead-poisoning. Alcohol, also, has considerable influence; sometimes, perhaps, by inducing the gouty habit. Some cases may be due to heredity, to chronic dyspepsia, or to climatic influences. Dickinson points out that renal diseases are more common in temperate climates, with their greater atmospheric variations, though these seem more likely to produce acute nephritis. There remains a good number of cases in which the cause cannot be clearly made out.

Symptoms.—The onset of chronic interstitial nephritis is generally quite slow, and marked by few distinctive features. Often, indeed, the kidneys are found to be granular in patients who die of other diseases, or one is struck down by cerebral hæmorrhage, without any symptom having attracted attention to the condition of these organs. Amongst early symptoms, which, occurring in a middle-aged person, should make one think of granular kidney, if not accounted for in other ways, are recurring or persistent headache, nausea and vomiting, shortness of breath, anæmia, loss of appetite, and general weakness. Often quite early it may be elicited that the patient is passing a large quantity of water, especially that he has to get up frequently at night to empty his bladder, and that the urine is pale and abundant. Occasionally no symptom may be sufficiently prominent until the sight is affected by renal retinitis, and the patient's eyes are examined with the ophthalmoscope.

The urine is quite normal at first; but in course of time it becomes abundant, pale in colour, almost watery, and of low specific gravity, 1005 to 1012. The solids, including the urea, are below the normal. The quantity of albumin is small, often a mere trace, generally not more than ·5 per cent. It may be for days entirely absent, or it is more abundant at one time in the day than another. The urine is quite clear, or throws down a very scanty deposit, in which a few hyaline or granular casts may be found. There is not at first any dropsy. Even after albuminuria has been recognised some time, there may be no more than a slight œdema of the ankles, or a little puffiness of the eyelids, or a watery conjunctiva. If the dropsy becomes considerable, either it has the characters of the cardiac form, and is the result of the secondary

implication of the heart; or it is a renal dropsy, and is due to the supervention of acute nephritis upon the chronic disease. The heart and pulse reveal the changes in the circulation that have been described. The impulse of the heart is displaced outwards, the aortic second sound is accentuated, and the first sound is perhaps reduplicated. The pulse is hard and cord-like; the arteries may be rigid and atheromatous.

In such a condition the patient may continue for several months or years, with varying health, but without any serious symptoms. But finally life is endangered by the several complications which have been indicated in the summary at the beginning of this chapter. *Cerebral hæmorrhage* carries off a certain proportion of cases. It may occur in the cerebrum, in the pons, or in the meninges, and results in apoplectic coma or paralysis of varying degrees, according to its extent and situation. *Pleurisy, pericarditis,* and *pneumonia* not unfrequently occur, and more rarely peritonitis. Uncontrollable *vomiting* is often the cause of death, and sometimes constant *diarrhœa.* The symptoms called *chronic uræmia* are more frequent than coma or convulsions.

The most important feature of chronic Bright's disease, from the clinical point of view, is its effect upon the heart. This organ at first hypertrophies, to overcome the resistance in the arterioles, but subsequently dilatation is added. As this becomes more pronounced a systolic murmur is produced at the apex either from eddies set up in the dilated ventricle, or from actual regurgitation through a widened mitral orifice, for which the valve has become insufficient. Finally, the heart becomes irregular in its action, other cavities are involved in turn, and a condition of inefficiency is produced identical with that of mitral valvular disease. Pulmonary congestion, perhaps with hæmorrhage, hepatic engorgement, ascites, and dropsy of the lower extremities follow upon this, and the urine is altered in the same manner as it is by a primary valvular disease—that is to say, it becomes scanty, high-coloured, and deposits urates, thus entirely losing the characteristics of the urine of granular kidney. The pulse may for some time retain its hardness, but eventually in some cases may become indistinguishable from the pulse of mitral disease. Finally, death may take place from cardiac failure.

Anatomy.—The kidneys are reduced in size, sometimes to a remarkable extent, and often one more so than the other; the two organs together may only weigh three or four ounces, and even less than one ounce and a half. The shape of the kidney is not materially altered, except from some irregularity in the rate of contraction in different parts. The capsule is adherent, and, if stripped off, carries with it small portions of renal tissue; the whole surface of the kidney is then seen to be covered with minute elevations (granulations) of $\frac{1}{16}$ to $\frac{1}{8}$ inch in size, with intervening depressions, and here and there may be cysts varying from $\frac{1}{8}$ to $\frac{1}{3}$ inch in diameter, and containing a clear fluid or colloid

material. The colour is mostly brownish-red, or dark red or pink, varying somewhat with the vascularity and the amount of interstitial tissue. The whole organ is tough; on section the cortex is found to be very narrow, sometimes reduced to $\frac{1}{4}$ or $\frac{1}{8}$ inch; the pyramids are also smaller than normal, and generally somewhat darker in colour than the cortex. Cysts may be present in the substance of the organ, and the divided vessels often stand out prominently on account of their abnormal thickness.

On microscopical examination it is seen that the anatomy of the organ is much altered. Here and there are patches infiltrated with leucocytes; these are most abundant in recent cases. In very old cases they have given place to irregularly distributed fibrous tissue, the contraction of which largely accounts for the small dimensions of the organ. The Malpighian capsules are thickened; many of the tufts are atrophied, and converted into fibrous nodules; and from the atrophy of intervening tissue the glomeruli are often crowded together irregularly.

In earlier stages there is some degeneration of the epithelium of the tubules, similar to that of the acuter forms, but slower in its course; and shed epithelium, leucocytes, and hyaline casts are seen in different tubes. Later the tubes become atrophied, partly by pressure of the new connective tissue, partly in consequence of the atrophy of the glomeruli, whose absence must deprive the tubes of their function, both by non-secretion of water and by the obstruction to the intertubal circulation which the atrophy of a glomerulus carries with it. The cysts arise from local obstructions to the tubes.

Pathology.—The inflammatory nature of this lesion is not universally accepted. Some regard it as a degeneration, beginning in the epithelium of the tubules and the glomeruli, and followed by secondary infiltration and contraction. Many look upon it as a change dependent upon and secondary to a general arterio-sclerosis: the arteries become thickened, obstruct the blood-supply, and hence lead to atrophy. Others hold that two forms of diseased kidney are included under the same name; one arising as just described, and hence to be called the *arterio-sclerotic kidney;* the other, the true granular kidney, in which the arterial sclerosis in the kidney precedes, instead of following, the change in the systemic arteries.

Diagnosis.—Granular kidney must not too hastily be assumed from the detection of a small quantity of albumin in the urine of a middle-aged person. The early symptoms must be carefully inquired into, the heart and pulse investigated, and the urine examined on several occasions.

The conditions generally considered to justify a diagnosis of chronic interstitial nephritis are a small quantity of albumin in the urine, associated with symptoms of ill-health, and evidence of cardio-vascular disturbance, such as high blood-pressure, thickened arteries, cardiac hypertrophy, retinal hæmorrhages, or retinitis, and œdema

of the feet. These signs are, however, not in themselves evidence of a granular kidney or of any renal disease. Persistent high tension may certainly exist independent of renal disease; it may arise apart from arterio-sclerosis, and arterio-sclerosis may occur without disease of the kidney. Allbutt has given the name *hyperpiesis* to a condition of independent high blood-pressure; and such high blood-pressure is attributable often to toxæmic states, possibly arising from the alimentary canal, among which gout, and the conditions associated with gout, may be included. If we admit this, it may be held to be unsafe to diagnose renal disease until more definite evidences of renal inadequacy or uræmia are manifest, such as headache, anæmia, nausea, lethargy, retinitis, convulsions, and coma. Practically, however, since increased blood-pressure undoubtedly does lead to arterio-sclerosis, and general arterio-sclerosis leads to a similar change in the kidney, these early indications of increased blood-pressure, especially if persistent, unrelieved by treatment, and accompanied by albuminuria and constant low density of urine, must warn us of the high probability of granular kidney, or chronic Bright's disease, either actually present or soon about to be.

In a more advanced stage of the patient's illness, it is often very difficult, or even impossible, to determine whether a case is one of *primary valvular* or *myocardial heart-disease* with albuminuria from congested kidneys, or one of granular kidney with secondary dilatation and hypertrophy of the heart. One must look for data in the history, the condition of the urine, the pulse, and the nature of any cardiac murmurs. It has already been said that the urine of chronic renal disease may in time assume all the characters of heart-disease; nor can the pulse be absolutely relied upon. A localised systolic murmur at the apex may occur in both cases; diastolic or presystolic mitral, and much more aortic murmurs, would be in favour of primary heart-disease. A mitral regurgitant murmur heard behind would be of doubtful significance. Previous attacks of rheumatism or of cardiac trouble from which the patient has temporarily recovered would be somewhat in favour of valvular lesions; but some cases of myocardial degeneration in alcoholic middle-aged persons with bad arteries are very difficult to distinguish from granular kidney.

In their typical conditions there can be no difficulty in distinguishing chronic *tubal* nephritis from cirrhosis of the kidney. In the former there are general dropsy, scanty urine, much albumin, and casts of all kinds; in the latter we see little or no dropsy, much urine, a small quantity of albumin, few or no casts, and there may be a very long history. The resemblance between red granular kidney and the white contracted kidney is closer: in the latter the albumin is abundant, and the patient is younger.

Prognosis.—This is unfavourable, but much improvement may take place, and life may be prolonged in some cases to an advanced age. The greater the extent to which the heart is implicated, the less is the expectation of life, and the occurrence of retinal changes is generally the indication of a short future.

Treatment.—After the removal of any condition which can be safely regarded as the cause of the nephritis—*i.e.*, alcohol, lead, constant exposure to cold, &c.—the objects should be (1) to diminish the call upon the excretory power of the kidney; (2) to reduce arterial tension, and the consequent strain upon the heart and vessels; (3) to remove anæmia; and (4) to treat special complications as they arise.

A quiet life, milk diet, or a diet largely free from purin-bodies, the use of diaphoretics, and occasional purgatives will fulfil the first indication. Complete rest is not practicable throughout the whole course, which may be many years, but it may be enforced when symptoms are serious, and at all times over-exertion and strain should be avoided. Similarly, at times of comparative health, or improvement, the diet may largely consist of milk, and farinaceous food, or even differ little from an ordinary diet, with this exception, that meat should be taken in moderation, and alcohol should be entirely stopped, or amount at most to a glass of claret or dry sherry daily. The patient should be warmly clothed, and residence in a genial climate is of great benefit.

The second indication is also fulfilled in the same way; but it may also be assisted by the use of nitro-glycerine ($\frac{1}{100}$ minim once or twice daily, and gradually increased) or sodium nitrite, or erythrol tetranitrate. These drugs reduce tension by dilating the arterioles, and often relieve the troublesome symptoms of headache and dyspnœa, which may occur in renal cirrhosis. Purgatives and diaphoretics both reduce tension, and assist in eliminating materials which are normally excreted by the kidneys.

Various preparations of iron should be given for the anæmia.

Among the complications which require treatment are uræmia, headache, vomiting, and cardiac dilatation and failure. The treatment of uræmia has been already given (*see* p. 920). For headache, antipyrin, caffein, and phenacetin are useful; pilocarpine injections may also be employed, or nitro-glycerine ($\frac{1}{100}$ minim), or one drop of liquor trinitrini. For vomiting, effervescing mixtures, dilute hydrocyanic acid, a few drops of tincture of iodine in water every hour, or a cold compress or blister to the epigastrium may be tried. For the cardiac symptoms, which result from dilatation and feeble contraction of the ventricle, digitalis should be given, the bowels should be kept open, and generally the case should be treated like one of heart-disease.

CONSECUTIVE NEPHRITIS

The cases of nephritis included in this group result, first, from various diseases of the ureter, bladder, and urethra, by which the passage of urine is interfered with; for instance, calculus, the pressure of tumours on the ureters, tubercular growth in the ureter, hypertrophy and thickening of the bladder obstructing the orifices

of the ureters, cystitis, and prostatic enlargement; secondly, from septic conditions of the same parts following upon stricture, cystitis, &c.

The following three forms may be recognised; they may, however, be associated together in the same kidney:

CHRONIC INTERSTITIAL NEPHRITIS

This is due to the increased pressure of urine behind an obstruction. If the obstruction is of long duration, the ureter and pelvis of the kidney become enormously dilated, and a hydronephrosis is produced, with more or less atrophy of the renal substance; but in the earlier stages the kidney becomes indurated from chronic interstitial change. This change is not infrequently seen in women after death from malignant disease of the uterus and vagina, which obstructs the ureter, but causes fatal symptoms too soon to allow the formation of a complete hydronephrosis. The kidneys are pale pink, or pale yellow, of about normal size, and very hard; on section there is some dilatation of the pelvis and the calices, and a little wasting of the pyramids, while the cortex is relatively broad. Under the microscope there is found infiltration of the organ with leucocytes, chiefly around the Malpighian capsules, and in the intertubal tissue; there is also some glomerular change, with a slight alteration of the tubal epithelium. In certain cases the process may go on to contraction of the new tissue, and the production of cicatrices.

The **Symptoms** are not prominent, and the condition of the urine is liable to be masked by the primary lesions of the ureter and bladder. Albuminuria is not always present; the urine is generally abundant and of low specific gravity. The disease affects one or both kidneys, according to the position of the obstructing lesion.

ACUTE OR SUBACUTE DIFFUSE NEPHRITIS

This is mainly an acute interstitial and glomerular nephritis, associated in some cases with inflammation of the pelvis of the kidney, or pyelitis. Both kidneys are affected. They are swollen, pale yellow, or yellow mottled with red, with prominent stellate veins. On section, the cortex is pale, or mottled, the pyramids often dark. Under the microscope, the intertubal tissue and the space between the Malpighian capsule and the tuft of vessels are seen to be crowded with round cells, but the tubules show comparatively little change, only some granular epithelium, with round cells which may have extravasated from the interstitial spaces. This form of nephritis is sometimes set up by operations about the urinary organs, or by washing out the bladder; in which cases the preceding condition of high pressure or of sepsis will obviously act as a predisposing cause.

The **Symptoms** begin suddenly with chill and rigor; and often there is moderate fever of intermittent type, the temperature being normal in the morning and from 99° to 101° in the evening. The urine is natural in amount, or abundant if there has been increased urinary pressure for some time previously. There is a small quantity of albumin, and there may be hyaline casts; but often these data are masked by the condition of the lower urinary passages—*e.g.*, the presence of cystitis. The general symptoms are weakness, languor, drowsiness, thirst, loss of appetite, nausea, and occasional vomiting. Improvement may take place slowly, or death from more acute nephritis, from suppurative nephritis, from exhaustion, or some intercurrent disease.

SUPPURATIVE NEPHRITIS

When suppuration of the substance of the kidney is secondary to lesions of the lower urinary passages, it is frequently associated with pyelitis, and hence is often termed *pyelo-nephritis*. All those causes which produce obstruction to the passage of urine, distension of the urinary passages, and cystitis, may ultimately result in suppurative nephritis; for instance, stricture of the urethra, prostatic enlargement and calculus; various diseases of the pelvic organs, uterus, ovaries, and appendages in the female, which involve the bladder or ureters; and paralysis of the bladder from spinal injury, spinal disease, or myelitis.

Anatomy.—The kidney is usually enlarged and softened. The surface is mottled pale yellow and red, and presents several small points of suppuration, some of which may be torn open when the capsule is stripped off. On section, the cortex shows the same mottled colour, the pyramids are usually red and congested, and the kidney has the appearance of one in a state of acute interstitial nephritis. The characteristic feature is the presence of numerous yellow streaks of pus, stretching in a radial direction from the surface inwards through the cortex, and even into the pyramids. They are sometimes wedge-shaped, or conical, with the base at the surface, in other cases simply linear. Under the microscope the kidney is seen to be acutely inflamed; there are numerous leucocytes in the intertubal tissue, some glomerular inflammation and swelling and nuclear proliferation of the epithelium of the tubes.

In most cases infection by micro-organisms spreads upwards along the ureter, pelvis of the kidney, and renal tubules. Sometimes infection is conveyed by the blood-vessels or lymphatics.

Symptoms.—It will be understood that the patient who suffers from this form of suppurative nephritis is generally already the subject of stricture of the urethra, enlarged prostate, cancer of the uterus, or of cystitis from retention of urine in spinal disease. The first symptoms are often chills or rigors, with a rise of temperature to 104°, 105°, or 106°, and there may be a regular

intermittent or remittent fever. Then follow the characteristics of a typhoid or septicæmic condition—loss of appetite, dry brown fissured tongue, nausea and vomiting, sometimes diarrhœa, sweating, and rapid emaciation. The patient is somnolent, in a more or less dreamy state, but without actual coma or delirium, till quite the last. The pupils vary, and have no special significance. The pulse is quick and feeble. Lumbar pain is sometimes present, and there is generally some tenderness on deep pressure in the loins. The state of the urine is determined by the preceding disease; it may thus contain pus, mucus, blood, or albumin; and it may be difficult to recognise any additional albumin, or even the hyaline casts, or the epithelial cells which result from an acute nephritis. In some cases, on the other hand, renal and pelvic epithelium and hyaline casts are found in sufficient quantity. The urine is often abundant, and the urea not much diminished; a considerable decrease may be due to preceding chronic nephritis. Morris notes that in cases with intermitting fever, the quantity of urine is often greater during the febrile periods than between them.

The duration of the illness is not more than four weeks, and may be as short as three or four days.

Diagnosis.—The occurrence of pyelo-nephritis in cases of disease of the bladder and ureters is generally signalised by the fever and the typhoid condition. It is likely to be confounded with pyæmia, other forms of septicæmia, enteric fever, peritonitis, and ague. In *pyæmia* one looks for secondary abscesses, which are absent in pyelo-nephritis. The temperature of pyæmia shows a more extensive range, and the rigors are followed by profuse sweating. Milder forms of *septicæmia* are almost indistinguishable from pyelo-nephritis; in the acute forms there are more restlessness and anxiety. Purpura and internal ecchymoses do not occur in pyelo-nephritis. In *enteric fever* the typhoid state is more slowly developed. Rose spots, typical motions, and the regular curve of temperature are in its favour; while rigors and an irregular fever would speak strongly for suppurating kidney. *Peritonitis,* which may arise from lesions of the bladder, is recognised by the absence of rigor, the greater pain, and more severe vomiting. In *malaria,* the intervals between the rigors are apyretic and almost healthy.

Treatment.—That suppurative nephritis may supervene should always be borne in mind in the treatment of diseases of the urinary organs; and it should, as far as possible, be prevented by dealing with its cause. When cystitis is the main difficulty, as in spinal cases, the bladder should be regularly emptied by the catheter, and irrigated with some antiseptic solution, such as salicylate of sodium (5 gr. to 1 oz.), quinine (1 or 2 gr. to 1 oz.), or borax (5 gr. to 1 oz.); and urotropin (10 grains), or helmitol should be given internally. When suppurative nephritis has declared itself, treatment is much less likely to be of use: but the employment of vaccines of the organism concerned should be considered. The

patient should be supported by nutritious but easily digestible food. The action of the skin should be promoted by vapour or hot-air baths. If there is much lumbar pain, hot sand may be placed on the loins, or dry-cupping may be employed. The bowels should be kept active. Internally, quinine, with small doses of opium or liq. morphinæ, seems the best remedy to give.

METASTATIC NEPHRITIS

Metastatic abscesses occur in *pyæmia* and occasionally in *malignant endocarditis* as the result of micro-organisms being conveyed by the blood-vessels to the kidneys.

Pyæmic abscesses are generally scattered through the cortex of the kidney; are of small size, more or less elongated, and sometimes conical; and are surrounded with a red zone of vascularity. Their occurrence does not obviously add to the symptoms of pyæmia. Albuminuria may occur independently of them, and cannot therefore be held to indicate their presence.

In malignant endocarditis, the abscesses may arise from the breaking down of embolic infarcts of various sizes. These are conical, but generally have a different shape from the abscesses of pyelo-nephritis, the base being relatively broader. Embolic infarction may be indicated by pain and by blood and albumin in the urine; but albumin occurring alone in the course of endocarditis does not of itself indicate suppuration.

Sometimes, both in pyæmia and endocarditis, there may be one or two large abscesses, instead of several small ones.

PERINEPHRITIS AND PERINEPHRIC ABSCESS

Perinephritis is the term used for inflammation of the cellular and adipose tissues around the kidney.

Ætiology.—It arises: (1) From injury—such as blows, kicks, or strains. (2) From the extension of inflammation from the kidney, the pelvis of the kidney, or the ureter. This may be the result of suppurative pyelitis, pyonephrosis, tubercle of the kidney, or calculus, which either sets up pyelitis, or itself ulcerates through the kidney or pelvis. (3) From inflammation, especially suppuration, in more distant parts spreading to the perinephric tissues; for instance, pelvic cellulitis, perityphlitis, abscess of the liver or spleen, caries of the spine and psoas abscess, or inflammation of the gall-bladder.

Pathology.—The perinephral tissue in different cases undergoes all the changes that may take place in other inflamed tissues. In early stages it is vascular, œdematous, and infiltrated; then points of suppuration occur, and finally one large abscess is formed.

The pus is free from odour, or very offensive; or it has a fæcal odour, from contiguity with the bowel. Sometimes shreds of gangrenous tissue are present. The kidney may be infiltrated or softened in the middle of the abscess. Occasionally the perinephral tissue is indurated by a more chronic process.

Symptoms.—These are to a certain extent those which accompany other inflammatory processes. The onset may be insidious, when there is nothing but some dull aching *pain;* in other cases it will be marked by rigor, with elevation of temperature, which continues uniformly high, or is intermittent in character. The pain is deep-seated, in the loin or side of the abdomen, and radiates to the hypogastrium, groin, or genitals. The pain in the loin is increased by pressure, and on bimanual examination a certain amount of fulness or resistance may be felt in that region.

As the case progresses, a more or less extensive *tumour* occupies the space between the last rib and the crest of the ilium, uniformly dull, bulging the flank, causing œdema of the loin, and perhaps fluctuating. The leg of the same side is often flexed at the hip-joint, and attempted extension causes pain; attention has also been called to the peculiar way in which a patient stands who has perinephritis, and this even when the inflammation has not reached the stage of abscess. The body is bent over to the affected side, the hip is a little flexed, and the hand rests on the same thigh. A certain resemblance to hip-joint disease is thus often assumed.

The urine is not necessarily affected; if the inflammation has resulted from ulceration of the kidney, pelvis, or ureter, pus from the perinephric abscess may pass into the urine (*pyuria*). In other cases albuminuria may occur from pressure of the abscess on the renal vein.

When pus forms, it is generally situated at first between the kidney and the lumbar muscles, and may make its way in various directions. If externally, it usually presents between the edges of the latissimus dorsi and the external oblique muscles; or it may pass downwards and point under Poupart's ligament. In other cases it opens into the colon, ileum or stomach; into the ureter, bladder, or vagina; or into the peritoneum, causing peritonitis. Or it perforates the diaphragm and sets up pneumonia pleurisy, and empyema; or, without perforation, it causes pleuritic effusions, or compresses the base of the lung by raising the diaphragm.

Diagnosis.—The conditions that are most likely to be mistaken for perinephritis or perinephric abscess, which is really a rare condition, are lumbago, spinal caries, cancer and tumours of the kidney, hydronephrosis and pyonephrosis, appendicitis, fæcal accumulations, splenic and hepatic tumours. The careful localisation of the lesion will distinguish it from spinal caries, hepatic and splenic tumours, and appendicitis. Fæcal accumulations, cancer of the kidney, and hydronephrosis are not accom-

panied by fever; nor is lumbago, and this is often a bilateral trouble. The lateral inclination of the body in standing is a useful guide, but hardly serves to distinguish it from hip-joint disease and psoas abscess.

Treatment.—This is mainly surgical. Local applications and opiates will relieve pain. If pus has formed it should be let out as soon as possible.

PYELITIS AND PYONEPHROSIS

Ætiology.—Inflammation of the pelvis of the kidney, or pyelitis, arises from several causes, of which the following have been recognised: (1) The action of turpentine and cantharides when given internally. (2) Febrile disorders, such as enteric and typhus fever, the exanthemata and pyæmia, as well as scurvy, diphtheria, and cholera. (3) It occurs, to a slight extent, in Bright's disease and diabetes; in the latter probably as a result of the irritation of the saccharine urine. (4) Many cases are due to a definite local cause, such as the irritation of foreign bodies in the pelvis and infundibula of the kidney, especially calculi and gravel, but also hydatids, blood-clots, and cancer. (5) Obstruction to the passage of urine may also lead to it by decomposition of the retained urine. (6) Inflammation may spread along the ureter to the pelvis in cases of cystitis and other forms of inflammation in the urinary passages, including those which result from infection of the urine with micro-organisms, such as the tubercular or typhoid or colon bacillus (*bacilluria*). It is probable that cold is sometimes a determining factor. The causes under the fourth, fifth, and sixth heads are much the most frequent, and it will be seen that these include the usual causes of suppurating kidney. Indeed, as already stated, pyelitis is constantly associated with that condition (*pyelo-nephritis*) or with cystitis (*pyelo-cystitis*).

Morbid Anatomy.—An acute or chronic form may be distinguished. In *acute pyelitis*, the mucous membrane is swollen, its vessels are injected, and the surface is covered with muco-pus; there are often small spots of hæmorrhage, and sometimes the inflammation takes on a diphtheritic form, patches of membrane adhering to the surface here and there. In calculous pyelitis there may be ulceration of the surface from the presence of the stone; and such ulceration may, as already indicated, lead to perforation and perinephritis. *Chronic pyelitis*, in which the membrane has a white or ash-gray colour, is often the result of long-continued obstruction; and accordingly there is at the same time dilatation of the pelvis, infundibula, and calices, with flattening of the pyramids, and more or less atrophy of the renal structure. As a result of the pyelitis, pus and urine may accumulate to such an extent as to form a perceptible tumour (*pyonephrosis*); and the fluid so retained may from time to time, through a change in the obstructing conditions (*e.g.*, shifting of a calculus), be discharged into the bladder,

so that the renal tumour subsides, and the urine suddenly contains a quantity of pus. Sometimes a pyonephrosis will open like perinephric abscess in various directions, such as into the loin, the iliac fossa, the bowel, the peritoneum, or the chest. In the renal pelvis there may be, besides pus and urine, blood, calculi, or other foreign bodies which have set up the mischief; and the urinary salts may be deposited, namely, urates in acid urine, and phosphates, if the urine is alkaline or ammoniacal, as it often is. The kidney is the subject of consecutive nephritis, either suppurative or interstitial, with more or less dilatation and atrophy. In some old cases the organ is so atrophied as to consist of little else than its capsule, and septa forming cavities which contain putty-like masses, the result of the inspissation of pus and the deposit of amorphous phosphates.

Symptoms.—In most cases the symptoms of pyelitis are combined with those of the lesions which have caused it. There is frequently some dull, aching pain in the loins, increased on pressure. The results of the inflammation generally show themselves in the urine. In early stages there are some mucus, a few pus cells, epithelial cells from the pelvis and infundibula, and perhaps blood. The cells of the pelvic and infundibular epithelium are mostly conical, pyriform, tailed, or fusiform in shape. The urine is acid, and contains a mere trace of albumin.

In later stages the urine contains pus in notable quantities.

Pyuria.—When such urine is passed it is turbid, and as it settles the pus forms a very pale yellow creamy deposit at the bottom of the glass, and mixes with the urine only just at the line of junction. The nature of this deposit can be determined by chemical tests, and by the microscope.

The usual *chemical* test is this: The supernatant liquid is poured off, and some liquor potassæ or liquor ammoniæ is added to the deposit; it quickly loses its colour, becomes translucent, and changes into a viscid, ropy liquid, which falls from vessel to vessel in a more or less coherent mass. If the urine should decompose and become alkaline within the body, the pus will undergo the same ropy change, and the urine will be mixed with this viscid, glairy fluid, instead of with creamy pus. This happens sometimes in pyelitis and pyonephrosis, if the urine retained in the dilated pelvis at length undergoes decomposition; and it happens frequently from the same cause in cystitis.

Ozonic ether effervesces when added to pus, and may be used as a test for its presence in the urine.

Under the *microscope* the creamy deposit shows numerous pus-corpuscles; but the epithelial cells of the pelvis are probably no longer produced in this stage.

The urine contains a very small quantity of albumin, derived from the liquor puris. It is often desirable to know whether the albumin present in such urine is solely due to pus, or is in part derived from diseased renal tissue. The pus should be separated

as completely as possbile by subsidence, or even by the use of the centrifuge; a large quantity of albumin must be due to something besides the pus. The presence of casts would, of course, suggest that the kidney structure was involved.

It has been already stated that the purulent urine may be retained by an obstruction of the ureter: during such retention, the urine in the bladder will be quite normal; and it will again become purulent when the obstruction is partially or wholly removed. In such cases a tumour forms in the flank, consisting of the pelvis and kidney distended with urine and pus, so long as obstruction exists. It is more or less rounded, or oval, in some cases lobulated; lies between the costal margin and the crest of the ilium; is dull behind, and generally has the colon in front or to its inner side. There may be a line of resonance between its dulness and that of the liver and spleen, but the areas of dulness are often continuous. The tumour is usually painful and tender. It disappears for a time, if the obstruction gives way.

There is generally some febrile reaction in pyonephrosis, and rigors sometimes occur. Micturition is frequent in most of the severe forms of pyelitis.

Diagnosis.—Pyelitis in its early stage must be distinguished by the local signs, and the presence of the characteristic epithelium in the urine. However, there is really a close resemblance between the epithelium of the renal pelvis and that of the bladder, and only a considerable predominance of caudate cells is in favour of pyelitis as against cystitis. When pus is contained in acid urine, it is more likely to come from the pelvis of the kidney than from an inflamed bladder. The diagnosis as to the presence of cystitis, and whether one or other kidney is involved, may be furthered by the use of the cystoscope or of the segregator.

Pyonephrosis may be confounded with the numerous swellings which occur in the right flank, and which have been alluded to under Perinephric Abscess. Abscess and hydronephrosis are those which are most likely to give difficulty. In the former there is more pain than in pyonephrosis, more severe fever, fluctuation is more superficial, and the preceding hardness is less defined. The skin may be œdematous, and the urine is often free from pus.

The **Prognosis** depends very much upon the primary cause. Pyelitis following fever or a mild cystitis is likely to recover. Pyonephrosis is a serious lesion; it may be fatal by perforation into the chest or abdomen; by exhaustion from continued discharge, or by the induction of lardaceous disease. Rarely the pus inspissates, and a cure results with the loss of one kidney.

Treatment.—The primary condition must be studied, and removed as far as possible (*see* Tubercle of the Kidney and Renal Calculus). Where the pyelitis is more or less distinct from its cause, and open to separate treatment, this consists, in acute cases, of rest; dry-cupping of the loins if there is hæmaturia or much pain; the free drinking of warm bland liquids, by which the urine

may be diluted; and the administration of salines (potassium citrate or acetate), with small doses of opium if there is much pain. When the case is more chronic and there is a free discharge of pus, astringents are commonly employed—namely, the mineral acids, alum, perchloride of iron, tannin, and acetate of lead; and, in very chronic cases, the balsams and allied drugs, oil of turpentine, oil of sandalwood, copaiba, benzoic acid, and cantharides, or direct antiseptics like salol and urotropin. In different cases vaccines of streptococcus or bacillus coli have been useful. A general tonic regimen may at the same time be desirable, a nutritious diet, fresh air or the seaside, quinine and cod-liver oil.

When the distended pelvis forms a tumour (pyonephrosis), as a rule the operation of *nephrotomy* should be performed, and the cyst opened; especially if there be constant pain, severe fever, and interference with the action of the stomach and intestine; or if the tumour is increasing in size, inflaming the surrounding tissues, or threatening to rupture. Under opposite conditions, and if the cyst empties itself into the bladder from time to time, palliative measures may be tried, such as rest in bed or on a couch, frequent hot baths, anodyne and emollient applications, gentle compression by belladonna plasters, and the avoidance of constipation and fæcal accumulation.

HYDRONEPHROSIS

By this term is meant the distension of the pelvis of the kidney by retained secretion; and the retention is, as a rule, the result of an obstruction in one or other part of the urinary passage, whether the ureter, the bladder, or the urethra.

Causes.—Hydronephrosis occurs at all ages, and is more frequent in females than males. It may be congenital—that is, in actual existence at the time of birth, when it may be so large as to constitute a serious obstacle to delivery; or it may develop after birth, although it is due to congenital causes; or it may be entirely the result of disease occurring in later life. Among the *congenital* causes are various abnormalities of the ureter, such as twists upon its axis, folds, reduplications, and valvular arrangements of the mucous membrane, contractions, or conversion into a fibrous cord. Sometimes the ureter joins the kidney at an acute angle or the opening into the bladder is thick and rigid, or a branch of the renal artery lies across its course. Another cause is an imperforate urethra. Congenital hydronephrosis is often associated with other congenital defects and malformations, such as club-foot, hare-lip, or malformations of the external genitals; and those affected with it are frequently still-born, or live only a short time.

The causes *in later life* are all those kinds of obstruction which have already been referred to—namely, in the ureter, impacted calculus, cicatricial stricture, cancer of the abdominal and pelvic organs, ovarian tumours, and peritoneal bands; in the bladder,

villous growths and cystitis with hypertrophy of the walls; and in the urethra, stricture and enlarged prostate.

Anatomy.—Any long-continued obstruction leads to distension of the parts behind it. If it is in the upper part of the ureter, the pelvis dilates, subsequently the pyramids of the kidney become flattened, and the kidney undergoes those changes of chronic interstitial (consecutive) nephritis which have been already described (*see* p. 929). If the obstruction is lower down—for instance, at the vesical orifice of the ureter—the ureter itself is involved in the distension. Such moderate degrees of hydronephrosis with consecutive induration of the kidney are common as the result of cancer of the uterus, vagina, and bladder in females. They, however, rarely lead to tumours that can be detected clinically, in many cases, no doubt, because death is brought about by the obstructive lesion too early for the extreme development of the hydronephrosis. But where the cause is congenital, or is less directly fatal in itself, such as calculus, or stricture of the ureter, time is allowed for the full development of the pressure effects of the retained urine upon the renal structures. The pelvis becomes distended, and the kidney more and more flattened out; and finally a large cyst is formed, capable of containing forty or fifty ounces, or even several pints of liquid, and consisting of a thin membranous sac, which may present here and there portions of the kidney substance, but in some cases is quite destitute of any trace of it. Inside the sac there are sometimes septa dividing it into separate cavities. If the ureter is involved, it may be enormously dilated, to the size, perhaps, of the small intestine or colon, or it may be entirely merged in the distended pelvis and kidney.

The fluid of a hydronephrosis varies with the amount of kidney-substance still remaining; generally it is equivalent to a very dilute urine; it is pale yellow in colour, contains a small proportion of urea, uric acid and salts, occasionally a trace of albumin, or a little pus. Urea and uric acid are absent in some cases, and the fluid consists mainly of water with a trace of sodium chloride. Pus in any quantity is not present unless there has been previous pyelitis, and the condition is then practically a pyonephrosis.

Hydronephrosis may affect one or both kidneys, according to the situations of the lesion or lesions causing it. In congenital cases it is frequently double—for instance, imperforate urethra must involve both kidneys equally; and the milder forms caused by cancer of the pelvic organs in women are often double. On the other hand, a calculus can only produce a single hydronephrosis, though it is possible that this cause may be in operation on both sides at the same time.

Symptoms.—A moderate degree of distension on one side, when the other kidney is healthy, may cause no symptoms whatever. If it is considerable, then a swelling is formed which becomes the prominent feature of the case, and has the usual characteristics of a renal tumour. It occupies one or other flank,

extending from the costal margin to the crest of the ilium, and reaching, according to its size, towards the middle line, or even beyond it. A cyst holding forty or fifty ounces may cause scarcely any prominence of the abdomen, but its presence will be detected by resistance to deep pressure, and by the difficulty of bringing together the two hands, placed one in front, the other under the last rib. With larger cysts, an unsymmetrical enlargement of the abdomen is produced, and the loin and flank are bulged; or in extreme cases there is uniform distension similar to that of ascites or ovarian tumour. Sometimes the cyst is limited to the upper part of the abdomen, and may resemble enlargement of the liver by hydatid. The tumour is smooth or lobulated, in some cases tense, in others quite flaccid; and fluctuation can be sometimes obtained. It is dull on percussion, the dulness reaching back to the loin, and forwards perhaps nearly to the umbilicus; the colon lies in front of it, and may cause a resonant note.

An important feature of the tumour of hydronephrosis is its liability to diminish in size suddenly, or even to disappear, from the escape of its contents into the bladder. Immediately afterwards, the patient has an abundant discharge of urine; and the cyst again slowly fills. Slighter variations in size or tenseness may occur, without any corresponding change in the urine being noticed. Local symptoms may be caused by the tenseness of the cyst, or its pressure on surrounding parts; such as pain, vomiting, dyspnœa, or interference with the heart's action. But these may be entirely absent. The urine in hydronephrosis is not much altered. Its quantity may be natural, since the healthy kidney compensates for the deficiency of its fellow. It may contain a trace of albumin, or a little pus: the urea and salts are in average quantity.

In cases of double hydronephrosis uræmia may occur from the retention of urinary constituents; the earlier indications of obstruction, when there is no tumour, are pains in the back or abdomen, partial suppression of urine from time to time, and increased frequency of micturition.

Diagnosis.—The tumour of hydronephrosis has to be distinguished from perinephric abscess, from pyonephrosis, hydatid of the liver or spleen, and, in extreme cases, from ascites and ovarian tumour. The history (*e.g.*, of calculus) may be the same in hydronephrosis, *pyonephrosis*, and *perinephric abscess;* the first is generally of longer duration, without severe, or even any, constitutional symptoms, whereas the other two conditions are likely to show evidence of suppuration. Perinephric abscess also gives the local signs of acute inflammation. In the absence of constitutional signs, pyonephrosis may be with difficulty diagnosed from hydronephrosis, and it may actually develop from it. *Hydatid* of the liver or spleen presses forwards or upwards, bulging the lower ribs, while hydronephrosis occupies the loin first; but I have seen a hydronephrosis due to calculus occupy the right upper quarter of the abdomen, just like hydatid of the liver. *Ovarian* tumour

should be recognised by the history of the enlargement, the position of the uterus, and the absence of the colon from the front of the cyst. In the rare case of a resemblance to *ascites*, the fluid withdrawn by paracentesis would contain urea and uric acid, and no albumin, or at most a mere trace, whereas in ascitic fluid albumin is abundant. In other cases also the aspirator and trocar may be used for diagnostic purposes. The spontaneous disappearance of the tumour, coincident with an increased flow of urine, is in all cases the strongest evidence of hydronephrosis.

Prognosis.—A simple hydronephrosis may cause little or no trouble for many years; the kidney may gradually undergo atrophy without the distension being such as to cause any serious trouble. The risk in such a case is that the other kidney may be at some time involved (*e.g.*, by calculous obstruction), and then death may be brought about by uræmia. If the cyst reaches a great size, or becomes very tense, it may rupture into the peritoneum, or it may press on adjacent parts—the stomach and diaphragm—and cause death by interference with nutrition, respiration, or circulation; occasionally, a cure results from the spontaneous discharge of the contents of the cyst, which never again collect. In the case to which I have alluded, the fifth or sixth subsidence in the course of two years was accompanied by the discharge of a calculus, which became impacted in the urethra, and was removed by incision.

In double hydronephrosis, death results from the primary cause (*e.g.*, cancer), or from uræmic symptoms.

Treatment.—Since recovery from hydronephrotic tumour sometimes takes place by the pressure of the liquid overcoming the obstruction, an attempt should be made to get the same result by friction and manipulation of the cyst. But sometimes it is too painful, or too tense, to make such a course desirable. It may then be aspirated. This should be done, on the left side, just at the anterior end of the eleventh intercostal space; on the right side, half-way between the last rib and the crest of the ilium, and two inches behind the anterior superior spine of the ilium. The fluid will probably accumulate again, and then the next step should be incision and drainage of the cyst. At this operation a way may possibly be found into the bladder, and the aperture dilated, or an obstructing stone may be sought for and removed. Sometimes this results in complete cure, the secretion ceasing and the wound closing; in other cases a fistula remains, which is often quite manageable. If the discharge is a serious trouble, or if it becomes purulent and threatens to exhaust the patient, the kidney must be excised.

LARDACEOUS DISEASE OF THE KIDNEY

The nature and ætiology of lardaceous disease have been already described (p. 785); phthisis, syphilis, continued suppuration, occasionally cancer and other cachectic conditions, produce the disease in the kidney as they do in other organs. But a slight amount of lardaceous change is found sometimes in cases of chronic nephritis, without any of the above causes being present; and it seems as if nephritis itself may be a local cause of the degeneration.

Morbid Anatomy.—In the kidney the lardaceous change affects the vessels first. The glomerular tuft is often first altered, then successively the vasa afferentia, the vasa recta, the vasa efferentia, and the intertubal vessels. In some cases however, the change can be found in the vasa recta before it is seen in the glomeruli. After the vessels, the basement membrane of the tubules is converted into the lardaceous material, but it is doubtful if the degeneration involves the epithelial cells of the tubules.

In the early stages of lardaceous change the kidneys present at first no appreciable difference, unless, perhaps, an undue distinctness of the glomeruli; but the application of iodine will bring out the vessels and Malpighian tufts by the dark brown or black colour (to the naked eye) which it gives them. If the change is very slight, it may be only seen with the help of the microscope in thin sections, stained as formerly described. In advanced stages the kidneys are much enlarged—it may be to twice their normal bulk. They have a whitish yellow colour, with some venules prominent on the surface; the capsule is slightly adherent. On section, the cortex is much widened, and has a pale yellow colour, more or less mottled with patches of white, while the pyramids are red or reddish-brown, forming thus a remarkable contrast to the cortex. Tincture of iodine stains the Malpighian tufts and their afferent arteries, and the closely packed vasa recta in the pyramids, so that the vessels look as if injected. On microscopic examination, it is seen that there is, in addition, much evidence of inflammatory change, to which the enlargement of the kidney is to be in great part referred. The tubal epithelium is swollen, granular, and fatty, the Malpighian capsules are thickened, and there are leucocytes extravasated into the intertubal tissue.

Sometimes the kidneys are almost normal in size, or even a little less, and somewhat rough or granular upon the surface.

Symptoms.—The symptoms of lardaceous degeneration commonly appear in patients already obviously suffering from phthisis, from tertiary syphilis with periosteal nodes, gummata, or necrosis, from caries, suppurating sinuses, discharging empyema, or similar lesions. Moreover, evidence of its involving the liver and spleen is sometimes, but not always, present when the kidneys are first called in question.

The first symptom is certainly sometimes the occurrence of albumin in urine otherwise normal—of average quantity, and good, even high, colour. With this there may be no other indication of renal disorder, only the evidence of the cause, and perhaps the changes in the liver and spleen. In other cases there is pronounced polyuria, and this may be the first change, even before the appearance of albumin (Dickinson). The urine amounts to seventy, eighty, or ninety ounces daily, it is pale in colour, of low specific gravity, 1008 to 1014, deposits little or nothing, and contains a varying, often small, amount of albumin. Paraglobulin is sometimes present in abundance, though this is not peculiar to lardaceous disease. In the deposit may be at most a few hyaline or granular casts, and, rarely, some that give the lardaceous reaction.

A third condition of the urine is seen in the last stage, when the lardaceous change is obviously complicated with nephritis; and the urine is scanty, highly albuminous, with numerous granular and fatty casts.

As to the cause of the alteration in the urine, it may be here enough to say that the degeneration of the walls of the vessels seems at once to account for the passage of water and albumin, as long as there is no great extent of tubal nephritis.

Next to the urinary changes, dropsy is the most frequent symptom. It has all the appearance of a renal dropsy, and there may be ascites and hydrothorax. Of its complications, pericarditis and peritonitis occur not infrequently, and may be fatal; but the other results of Bright's disease—namely, cardiac hypertrophy, high arterial tension, retinitis, hæmorrhages, and uræmia—are quite rare, though it appears that they do sometimes occur. Diarrhœa may occur from co-existing lardaceous disease of the intestine. Death takes place from increasing dropsy, exhausting diarrhœa, serous inflammations, or from the effects of the original disease.

Diagnosis.—If there is a sufficient cause, such as phthisis, syphilis, or suppurating lesion, and evidence of lardaceous change in the liver and the spleen, which are generally enlarged, smooth, and hard (though they may be considerably diseased without increase of size), then the presence of albumin in the urine speaks strongly for lardaceous degeneration of the kidneys. All the more is this the case if the urine does *not* present the features of ordinary nephritis—that is, if it is of normal quantity and colour, or if, being abundant, it has a larger quantity of albumin than is common in granular kidney. If the condition of the urine is compatible with an acute or subacute tubal nephritis, lardaceous disease can only be inferred from the presence of an exciting cause and the evidence of the change elsewhere.

Prognosis.—This is decidedly unfavourable, especially if the stage of dropsy is reached. But in earlier stages, and in cases where the causative condition can be more or less controlled—for instance, tertiary syphilis by iodide of potassium, diseased joints and necrosis by suitable operations—the condition may last for

some time without causing grave symptoms, and even it seems possible that practical cure may result.

Treatment.—This consists in removing the cause so far as it can be done. Beyond this the administration of potassium iodide, of cod-liver oil, and of mild preparations of iron, such as the syrup of the iodide, seems to be attended with some benefit. Complications must be treated in the same way as in the different forms of Bright's disease.

TUBERCLE OF THE KIDNEY

Tubercular disease of the kidney occurs in two forms: (1) Primary disease, formerly known as strumous pyelitis or pyelo-nephritis, and scrofulous pyelitis. (2) Secondary or disseminated gray tubercle.

PRIMARY TUBERCULOSIS OF THE KIDNEY

Ætiology.—This is obscure beyond the fact of its association with other tubercular lesions, and its probable dependence upon the same causes (hereditary influence, cold, deficient hygienic surroundings, &c.); but its localisation in the kidneys cannot always be explained. It affects men more often than women, and occurs at all ages, but is least common in quite young children.

Morbid Anatomy.—The first change is the deposit of tubercles in the substance of the kidney, either cortex or pyramids. When first seen these are generally already yellow and cheesy; they enlarge, run together, break down into abscess cavities, and ultimately open into the calices and infundibula. But it is an important feature in the history of primary tubercle that the kidney is not alone affected; the disease commonly affects at the same time more or less the urinary passages, and, it may be, every one of the urino-genital organs. Tubercles are deposited in the mucous and sub-mucous tissues of the pelvis of the kidney, which ultimately break down, leaving ragged ulcers opening into the pelvis, and discharging pus, blood, tubercular *débris*, and portions of connective tissue into the current of the urine.

In extreme cases the kidney is almost entirely destroyed, so that scarcely any renal tissue remains. This is caused partly by the spread of the caseating process, and partly by the distension of the pelvis, in consequence of the ureter being obstructed. Thus, what has not been destroyed by tubercular ulceration is converted into dense fibrous tissue, and forms septa, separating the several abscess-cavities from one another; and such cavities may ultimately come to be lined with a smooth membrane. Their contents are a semi-fluid cheesy material, or a putty-like mass, which contains an abundance of calcium salts. The capsule is thickened, and may be even like fibro-cartilage.

The *ureter* is similarly diseased; its wall is thickened and rigid from tubercular deposit, which finally ulcerates. The thickening

may be sufficient to obstruct the canal of the ureter, or the latter may get blocked by fragments of tubercular matter, or by coagula of blood or pus coming down from the pelvis. If such an obstruction is at all complete, the urine and diseased products are retained in the pelvis of the kidney, distension takes place, and a pyonephrosis is the result.

The *bladder* is affected very like the ureter, either at the same time as or even before the kidney. Tubercle is deposited in the submucous tissue, and eventually the mucous membrane is covered with cheesy deposit, and much ulcerated. Sometimes the disease spreads into the *urethra*, and in men the *prostate, vesiculæ seminales*, and *testes* may become involved. The genital organs of women are much more rarely diseased under corresponding circumstances.

The disease is at first unilateral, but the kidney of the opposite side may be infected from the bladder. This extension, however, may be long delayed, or, indeed, may never occur. Another possible result is a general tuberculosis, with its inevitably fatal ending.

Symptoms.—In most cases the symptoms are those of pyelitis and cystitis combined, or pyelo-cystitis. There is more or less dull pain in the loins, which may be paroxysmal in character; and there is often some tenderness on pressure. Severe colicky pains occur if the ureter becomes plugged by tubercular *débris*. The kidney is not infrequently so much enlarged by tubercular deposit, or by distension of the pelvis and calices, that it can be felt as a tumour in the flank.

The condition of the urine is most important. It is generally acid, with a more or less abundant deposit of pus, in which may be found pelvic and vesical epithelium, shreds of connective tissue, and *débris* of tubercle; while tubercle-bacilli can be demonstrated if the urine be centrifuged and the deposit suitably stained, or their presence can be proved by cultivation or animal inoculation. Blood is often present from time to time, but not generally in large quantity. Albumin occurs in proportion to the amount of pus. Tube casts are rare. The amount of urea will depend on the opposite kidney, which may enlarge enough to eliminate the normal amount Sometimes the urine is ammoniacal and ropy, from retention and decomposition in the pelvis; sometimes from the co-existing cystitis. With cystitis, also, micturition becomes frequent and often painful.

Fever is generally present at some period of the disorder, and eventually becomes persistent, with morning remissions and high evening temperatures. As the disease progresses, emaciation, loss of appetite, and prostration become marked. In many cases other organs—such as the lungs and intestines—are involved. The opposite kidney also may be affected with the same lesion, or with lardaceous disease; and there is a gradual diminution in the excretion of urea, sometimes with scanty, occasionally with abundant, secretion of urine. Death takes place from the exhaustion of prolonged suppuration or the tubercular fever, from

pulmonary or intestinal lesions, or from uræmia when the second kidney is seriously involved.

Diagnosis.—A tuberculous kidney is most likely to be confounded with renal *calculus*, since they may both cause severe renal colic. Hæmorrhage is more abundant in the latter, pus more continuously present in the former. In all cases of turbid or purulent urine, in which tubercle is possible, the urine should be examined microscopically and bacteriologically for evidence of the bacillus. Often the family history, the previous history of the patient, the co-existence of pulmonary phthisis, or other tubercular lesion, such as tubercle in the epididymis, suggests or confirms the diagnosis. Sometimes the ureter is so thickened by tubercle as to be felt through the abdominal organ as a hard rigid cord. Palpation of the kidney cannot be relied upon, but the cystoscope may give valuable information both as to the presence of tubercle and the condition of the other kidney. By this means the mucous membrane of the bladder around the orifice of the ureter on the affected side is seen to be reddened, swollen, and abraded or ulcerated; and miliary tubercles may be seen scattered in the neighbourhood: while a turbid urine may pass from the orifice in a sluggish stream.

Prognosis.—The prognosis of tubercle of the urinary organs is like that of the lung, dependent on the virulence of the infection, the resistance of the patient, and the promptness and thoroughness of treatment. As recent methods of research render a diagnosis possible at a much earlier stage than formerly, the outlook of the patient is proportionately more favourable.

Treatment.—The strength of the patient must be supported by good food, and by tonics, such as iron, quinine, cod-liver oil, and extract of malt; and opiates and local applications should be used to relieve the pain. In early stages the use of tuberculin at the same time that the opsonic index is watched (*see* p. 539) has led to relief of symptoms and delay in the progress of the disease. But pronounced local conditions will call for an operation either to let out pus, or to excise the organ entirely. This is generally the best course to take, since the tuberculosis is often limited to one side, and even if the ureter and bladder are involved, the ureter may be also removed, and the bladder may recover. Moreover, if the cystoscope shows characteristic changes around the vesical orifice of the ureter, the kidney is probably too much damaged to escape destruction (Symonds). Removal is out of the question if the kidney of the opposite side is extensively diseased, or if tubercle has taken firm hold of other viscera; or if the patient is much exhausted. A small abscess in the kidney may be incised and drained, with some hope of ultimate success; and for large collections of pus in connection with obstructed ureters, nephrotomy is also valuable by relieving tension and pain, and giving direct exit to the purulent secretions.

SECONDARY TUBERCULOSIS OF THE KIDNEY

This occurs as a part of acute general tuberculosis. The liver, spleen, and lungs are often at the same time affected. The tubercle appears in the form of minute gray or yellow deposits, one or two millimetres in diameter, scattered irregularly, and as a rule rather scantily, in the cortex and medulla of the kidney. A few may be seen on the surface, and others are revealed by section; they are round in shape, or slightly elongated in the direction of the tubules. They present the characteristic minute anatomy of tubercle. The rest of the kidney is healthy, and, as a rule, there are no clinical symptoms attending their deposition. Albuminuria may occur, and Roberts mentions a case from a French source, in which an unusually abundant deposit of tubercular granulations caused violent lumbar pains, with strong contraction and exquisite tenderness of the lumbar muscles.

PARASITES IN THE KIDNEY AND URINARY ORGANS

The parasites invading the urinary organs are the *Echinococcus hominis* (a *hydatid*), the *Bilharzia hæmatobia*, the *Strongylus gigas*, and the *Pentastoma denticulatum*. The last two are exceedingly rare, and need not here be described.

HYDATID OF THE KIDNEY

The life-history of the Echinococcus hominis and the development of hydatid cysts have already been described among the diseases of the liver. Hydatid cysts are very rare in the kidney; they form either in the substance of the gland, or between it and its capsule, and they grow to a variable size. They undergo the same changes as they do in the liver, and lead to corresponding local difficulties. As the cyst grows it gives rise to a tumour, which is generally globular and tense, and exerts considerable pressure on surrounding parts. Not infrequently it ruptures into the pelvis of the kidney, and the daughter-cysts, either whole or in fragments, escape and are discharged with the urine, if they are small enough to pass down the ureter. The cyst may rupture into the intestine, or it may, after compressing the diaphragm and the base of the lung, open into the bronchi. Such ruptures may occur spontaneously, or be brought about by a blow or other injury. In other cases the cyst suppurates, or it becomes converted into the putty-like remains which have been previously mentioned (*see* p. 791).

Symptoms.—One of the symptoms, and it may be the only one, is the presence of the tumour caused by the cyst. It is situate in

the loin, with the colon in front of it; it is generally more or less globular and tense, and occasionally, but not always, gives the so-called hydatid thrill on percussion. The cyst may not be large enough to be detected, and may rupture in its early stage. If it bursts into the pelvis of the kidney, the urine will contain daughter-cysts, or shreds of them, or a milky detritus, in which the characteristic hooklets may be found. The cysts, or any portions of them, may become impacted in the ureter, causing renal colic; or in the urethra, after passing through the bladder. If rupture takes place into the intestine or bronchi, cysts, or portions of them, will be got rid of by these passages, or the discharge of them may soon cease, and recommence after a longer or shorter interval. Pyelitis and cystitis may result from the passage of hydatids; but if no rupture takes place the urine is quite normal. Suppuration of the mother-cyst produces an abscess, which becomes manifest by increasing pain, tenderness, the implication of the surrounding tissues, and characteristic fever.

The **Prognosis** is fairly favourable, since a free discharge of the cyst and its contents is so often possible, and, as a fact, a good many recoveries have been recorded. The duration of hydatid of the kidney is variable, and may be as long as even thirty years.

The **Diagnosis** depends upon the presence of a renal tumour, combined with the discovery of cysts or hooklets in the urine. This latter is not in itself conclusive as to its position in the kidney, since a hydatid cyst behind the bladder may rupture into it. In doubtful cases a rectal examination should be made. A history of renal colic accompanying the discharge of cysts or fragments would be strongly in favour of their origin in the kidney. The tumour formed by the hydatid is most likely to be confounded with *hydronephrosis;* this may sometimes be distinguished by the typical variations of size; for, though a hydatid may also empty itself, this will be most likely accompanied by the appearance of daughter-cysts, or scolices and hooklets, in the urine. But the resemblance may be so close as only to be solved by the use of the exploring needle, when the hydronephrosis will yield a urinous fluid, the hydatid a clear or opalescent fluid with a minute quantity of salts and a trace of albumin, and perhaps under the microscope some scolices and hooklets.

Treatment.—The withdrawal of the fluid by aspiration has yielded good results in the case of the kidney, as it has in the case of the liver. But the more certain method of cure is to cut down upon the tumour, through the loin if possible, to empty the cyst by tapping, and then incise it, and stitch the membrane to the edges of the wound in the parietes. If the contents of the cyst are being discharged by the bladder, this operation is not necessary. Impaction of cysts in the ureter requires the treatment proper to renal colic: opium, hot baths, and soothing local applications.

BILHARZIA HÆMATOBIA

In different parts of Africa (Egypt, Natal) the inhabitants are liable to a form of endemic hæmaturia, which is due to the invasion by a parasite of the mucous membrane of the urinary passages. This parasite, called *Bilharzia,* from its discoverer Bilharz, belongs to the order *Distomidæ.* It is elongated in shape, the male being about half an inch and the female three-quarters of an inch in length. In congress, the female lies in a groove on one side of the male, called the gynæcophoric canal. The ova are egg-shaped bodies, $\frac{1}{170}$ inch in length, and $\frac{1}{400}$ inch in thickness, and present at one end a sharp spine, which is sometimes quite at the extremity (terminal), sometimes a little distance from it (lateral). The embryo is a ciliated body, which can be seen moving within the ovum, and, when liberated, glides rapidly about by the action of its cilia, and with various contractions of its body.

The parasites and the ova have been found in the minute veins of the bladder, ureter, pelvis of the kidney, uterus and rectum, and in the portal vein and its tributaries; and, as a consequence, extensive inflammatory changes occur in the mucus membrane and submucous tissues of the bladder, ureter, pelvis, and rectum. The mucous membrane of the bladder, especially posteriorly, presents patches from a quarter to one inch in diameter, which are swollen, vascular, ecchymotic, and covered with tough mucus or yellowish exudation; or there are warty prominences encrusted with urinary salts. The ova of the bilharzia are found in great numbers in the mucus and exudation on the surface, and in the mucous and submucous tissues; and the parasites themselves lie in smooth-walled spaces, which are, no doubt, altered veins. In the ureter similar changes occur; its wall is much thickened, and the swelling causes obstruction to the passage of the urine, and may lead eventually to pyelitis and pyonephrosis. In some cases the dilated ureter shows a number of small retention cysts in its mucous membrane. The parasite directly affects the pelvis of the kidney much less commonly. The ova may do harm in another way—namely, by serving as a nucleus for urinary calculi, which, indeed, appear to be quite common in Egypt. The liver is generally enlarged and presents a quantity of new fibrous tissue, in which numerous ova are found. In its most characteristic form, the dense white fibrous tissue is localised around small branches of the portal vein, while the hepatic tissue generally is of gray-brown colour, and relatively little involved. There is no contraction of the organ as in alcoholic cirrhosis.

Very remarkable changes take place in the bowel. In the colon are seen numerous polypoid growths or adeno-papillomata, sessile or pedunculated; and these may be accompanied by a diffuse colitis. The rectum is sometimes almost blocked by similar adeno-papillomata, which may be mistaken for piles; the mucous membrane is much thickened, and beneath it are large hæmorrhages.

With regard to the entrance of the parasite into the system, it has been suggested that the embryo is contained in dirty water in which the natives bathe or paddle, and that it enters either by the skin, or the urethra, or the rectum; or that it is contained in drinking water; or that, as with the tapeworm, the embryo passes through some intermediate host, and that this, a mollusc perhaps, is eaten with the food.

But the balance of opinion is in favour of the view that entrance is effected through the skin, and probably from infected water in which the sufferer bathes or paddles, or from wet soil in which he walks barefoot.

Symptoms.—These are hæmaturia, sometimes pain in the loins or perinæum, and, in severe cases, anæmia, from loss of blood. The hæmaturia is mostly of vesical origin; the urine is passed clear, and is followed by about a teaspoonful of blood. The urine may at times contain no blood, but only whitish flocculent matter, and shreds and filaments of mucus in which the ova of bilharzia are found in great numbers. There is often a trace of albumin. Micturition is more frequent, and there is pricking at the prepuce or root of the penis. In later stages more pronounced cystitis is present. If the rectum is involved, there is a discharge of mucus with straining, and later prolapsus ani.

The symptoms may be of temporary duration, especially in boys, and in many cases, though the disease persists for years, there is little suffering. On the other hand, the loss of blood may lead to a high degree of anæmia; and septic cystitis, pyelonephritis and pyonephrosis, or sloughing of the rectum and septicæmia, may bring about death.

Calculi in the bladder and urinary fistulæ are also serious complications.

Treatment.—Sandwith regards liquid extract of male fern as the only remedy of any value in bilharziosis. It diminishes hæmaturia and vesical irritation, and lessens the number of ova passed. The dose is 15 minims three times a day; but it must not be continued for more than fourteen days at a time, or it may cause unpleasant symptoms. The cystitis may be treated with antiseptic irrigation (boric acid), or by salol, urotropin, or helmitol internally. Polypoid growths in the rectum should be removed.

Prevention.—The drinking-water should be from a pure source and should be filtered; uncooked vegetables, fresh-water fish, and molluscs should not be eaten; and bathing in fresh water should be prohibited.

NEW GROWTHS IN THE KIDNEY

The following tumours are met with in the kidney: Adenoma, fibroma, forming small round nodules in the pyramids, leuchæmic deposit, masses of lymphadenoma in Hodgkin's disease, cavernous angeioma, papilloma, sarcoma, and epithelial carcinoma. Of these the most common are sarcoma and carcinoma, and it is only these, as a rule, that are large enough to become of clinical importance.

SARCOMA

This is generally primary, and frequently occurs in quite young children or infants. The organ is enlarged to an immense size, often filling half the abdomen. It presents the usual characters of a renal tumour, filling out the loin, but increasing downwards and inwards towards the umbilicus, having the colon in front of it, and rounded or oval, with no sharp edges or notches. It may be composed of round or of spindle cells; and its consistence varies, sometimes being hard, at others so soft as to invite exploration for fluid. The tumour grows rapidly, causes neither pain nor hæmorrhage, but kills finally by exhaustion and emaciation. Bland Sutton points out that many of the sarcomatous tumours of this region arise in the adrenals, and probably a few in accessory adrenals. This is especially the case in adults, in whom the adrenal tumours are unilateral, whereas in infants they are often bilateral. The rena sarcoma of adults is accompanied by pain and hæmorrhage; but the adrenal tumour does not cause bleeding. The importance of the distinction is that removal of adrenal sarcomata is much more likely to be successful than that of renal growths.

EPITHELIAL CARCINOMA

This is primary or secondary. *Secondary* cancer occurs occasionally in the course of cancerous disease of other organs, such as the female genitals, the rectum, other abdominal viscera, or the female breast. The nodules are small, and their presence is not generally productive of special symptoms. As a rule, both kidneys are involved. *Primary* carcinoma occurs mostly in persons of middle or advanced age, and is more frequent in men than in women. It is less common than secondary carcinoma. The cause of primary cancer of the kidney is not, as a rule, to be ascertained. Sometimes it appears to have followed a blow, or it is found in association with calculi; and it may be assumed, as in the analogous case of hepatic cancer and biliary calculi, that the calculi have acted as a constant source of irritation. It mostly affects one side only. It causes considerable enlargement of the kidney, but rarely to the same size, proportionately to the body, as does sarcoma in children. Encephaloid growths are most common, and colloid change frequently

occurs, but scirrhus is rare. Hæmorrhage and softening often take place, as in cancers elsewhere.

Histologically, two varieties of carcinoma can be distinguished, one in which the cells are relatively small in size with a large nucleus, and a protoplasm darkened by numerous granules; the other in which the cells are large, the nucleus small, and the protoplasm clear, apparently from its containing fat, which has been dissolved out in the preparation of the specimen. Tumours of this last kind, from the close resemblance of their elements to those of the fasciculate layer of the suprarenal cortex, have been called *hypernephroma*, and they are believed to arise from suprarenal remains in the substance of the kidney. They are often encapsuled, and present on section irregular division into lobules by fibrous tissue, with scattered areas, some of bright yellow colour, others of red or brown colour, in addition to cysts the walls of which may be calcified. The tumour cells vary in size, often reaching the dimensions of giant-cells, and have a tubular or alveolar arrangement, or are grouped radially round vessels. The suprarenal origin of these tumours is not universally admitted, and many think that some at least of them are epithelial growths from the cells of the convoluted renal tubules.

Symptoms.—These are tumour, hæmaturia, and pain. The characters of the *tumour* have already been in part indicated; it may be irregular or nodulated; its mobility may be limited by adhesions; manipulation will elicit tenderness. Rarely the tumour pulsates, and a *bruit* may be heard in it. *Hæmaturia* is frequent; it is intermittent, variable in amount, but generally moderate. Albumin does not occur without blood, unless there is independent Bright's disease. Cancer-cells are sometimes recognised, but they may be lost among blood-corpuscles; or epithelium from the bladder or pelvis may be mistaken for them: and on the whole they cannot be relied upon for a diagnosis. Apart from blood and cancer elements, the urine may be perfectly normal in quality, density, and colour. *Pain* is variable, and often severe. It is situate in the loin and hypochondrium, or shoots down the groin to the thigh; it is not associated with retraction of the testicle. If blood coagulates in the pelvis of the kidney, the passage of the clots by the ureter may be accompanied by the pain of renal colic (*see* Renal Calculus). Extension of the growth to the lymphatic glands may lead to pressure upon the roots of the spermatic veins and the production of a *varicocele*, or *œdema of the legs* may occur.

The remaining symptoms of cancer of the kidney are anorexia, nausea, vomiting, irregularity of the bowels, either constipation or diarrhœa, and progressive emaciation and anæmia, as in other malignant affections. The duration varies with the kind of cancer—it may be from six months to two years, or even more. Death takes place commonly from exhaustion: but the growth may spread to the spine, and cause paraplegia; to the peritoneum and viscera, and cause local symptoms; it may rupture into the peritoneum; or there may be profuse bleeding into the tumour itself. Finally,

secondary deposits in the brain may assist the fatal end. Hypernephroma very commonly leads to metastases, in the lungs, liver, bowels, or lymph-glands, and especially in the bones and muscles.

Diagnosis.—Cancer of the kidney may be recognised by the co-existence of pain, hæmaturia, and tumour of the renal region; but, in the absence of hæmaturia, the nature of the tumour has to be very carefully investigated. It has to be distinguished, first, from enlargements of other organs; secondly, from other diseases of the kidney. From enlargement of the *liver* it is distinguished on the right side by the presence of a band of resonance between its upper margin and the ribs; if it is adherent to the liver, the edge of that organ may still often be felt on the anterior surface of the tumour. The presence of bowel (colon) in front of the tumour is also characteristic of its renal origin. The same holds good for the left kidney as compared with the *spleen;* and the latter is recognised by its sharp notched edge, its smooth surface, and its generally uniform enlargement in a downward and inward direction. An *ovarian* tumour grows up from below, whereas a renal tumour begins above and grows downward. *Mesenteric glands* lie nearer the middle line than the kidney, and form very nodular masses; isolated enlarged glands may give a clue to the nature of the larger mass. If cancer of the *colon* simulate renal cancer, intestinal symptoms are mostly present. *Accumulated fæces* on the left side would show more variability in size and consistence, and the diagnosis can be cleared up by the use of an enema. *Perinephric abscess* gives infiltration of the skin, local pain and tenderness, and febrile reaction. The development of varicocele in an elderly man should lead one to examine the loin for a tumour; and the association of a tumour of a bone with hæmaturia should suggest the existence of hypernephroma.

The diseases of the kidney which may resemble cancer are pyonephrosis, hydronephrosis, hydatid, cystic degeneration, and tubercular disease. From the first three it should be distinguished by its solid feel; but a cancer may exceptionally be very soft, or a renal cyst may be small and tense, so as to give no certain sense of fluctuation. A tense uniform globular surface would be in favour of a cyst, but a lobulated hydronephrosis may be mistaken for new growth. By the time that *tubercular disease* causes enlargement enough to resemble cancer, it is practically a pyonephrosis. *Cystic* disease commonly involves both kidneys, and has not the other local signs of cancer; the pale abundant urine, with a trace of albumin, should distinguish it. In cases of doubt as between cancer and cystic enlargement, the aspirating needle may be safely used; and in any case Röntgen rays may assist the diagnosis.

Treatment.—If cancer of the kidney is recognised early enough, its removal by operation should be attempted, otherwise the treatment must be mainly palliative; the objects being to relieve pain by opium and local applications, to keep up the strength by an easily digestible diet, and the moderate use of wine or small quantities of brandy.

CYSTS IN THE KIDNEY

The following forms of cystic disease of the kidney occur: (1) Small cysts in granular kidneys already described; (2) Extensive cystic change known as *cystic disease* or *polycystic kidney*; (3) Simple cysts; (4) Dermoid cysts, which are exceedingly rare, and have the same characters as elsewhere; and (5) Hydatid cysts, which have been dealt with under Parasites in the Kidney.

CYSTIC DISEASE

(*Polycystic Kidney*)

This occurs in adults and in the fœtus, and leads in both cases to an enormous enlargement of the kidneys, which in the majority of cases is bilateral. In adults, the organs weigh from one to five or six pounds, and may be detected during life as tumours occupying the lumbar region. They are more common in males than in females, and the patients are mostly of middle age or older.

Pathology.—On section, the kidneys are seen to consist almost wholly of cysts of various sizes, containing a fluid which is clear, or turbid, yellow, pink, red, or purple, sometimes viscid, colloid, or purulent. Always albumin, and sometimes blood-discs, leucocytes, and cholesterin are found in the fluid, but urea and uric acid are generally absent. The cysts are surrounded by fibrous tissue, in which only remnants of renal tissue can be found; they are lined with epithelium. The pelvis, ureter, and bladder are healthy, or the pelvis may be dilated. The pathology of these kidneys is still open to doubt. There is no essential difference in structure between those which are congenital, and those which are first discovered in adults at the age of fifty or more. In the adult cases, the cysts have all the appearance of having arisen from renal tubules which have been dilated as a result of obstruction by fibrous tissue; and they are often regarded as an extreme development of the cyst formation which occurs in granular kidney, although Gombault and Hommey observe that the sclerosis exists only around the large cysts, and is absent from the smaller cysts—in other words, that the sclerosis is secondary. But it is less easy to understand than the origin of a chronic interstitial cyst-forming nephritis in the fœtus. For these cases of *congenital cystic kidney*, Koster in 1860 suggested a fault in development, and the view of Shattock has been widely accepted that they are due to a fault in the development of the kidneys; that the blastema from which the kidneys arise is not properly differentiated from that of the Wolffian bodies; that these bodies are mixed up, as it were, with the kidneys, and give rise to the cysts. But the fact that congenital granular kidneys have been recorded rather supports the view of their identity with the adult cases.

Symptoms.—In congenital cystic disease the kidneys may occupy the greater part of the abdomen, and press upon the diaphragm; and death may happen *in utero*, or during birth; or the child may survive a few months, or in the event of the disease being unilateral, a few years, when at length death may result from uræmia. The change is often associated with other congenital malformations, both of the urinary organs and other parts.

In adults the symptoms are often very obscure, but bear a general resemblance to those of chronic interstitial nephritis. The urine is generally abundant, of low specific gravity, pale in colour, and contains a little albumin and granular casts. Sometimes there is hæmaturia. Hypertrophy of the heart and high arterial tension have been observed. There may be lumbar pain, but dropsy is generally absent. In a patient under my care, there was daily intermitting pyrexia for several weeks preceding death. The termination is often by uræmic coma or convulsions; or there is cerebral hæmorrhage, suppression of urine, or pulmonary complication, such as bronchitis, œdema, or pneumonia.

For **Diagnosis** one must rely upon the presence of enlarged kidneys (for the change is generally double), associated with the characteristic urine, and cardio-vascular changes; but the disease is quite rare.

Treatment must resemble that of chronic Bright's disease.

SIMPLE CYSTS

Cysts of considerable size are sometimes found. They may be three or four inches in diameter, and exceptionally very much larger. They arise from the cortex, and project on the surface. Their contents are a clear limpid or gelatinous fluid, containing a little albumin and some salts, but no urea or uric acid. The remainder of the kidney may be quite healthy. Such cysts may be unrecognised during life; if very large, they form tumours which may require to be tapped and treated surgically on the same principles as hydronephrosis.

MOVABLE KIDNEY

The name *movable kidney* is given to one that is readily displaced from its normal position, and can be moved more or less freely in the abdomen. This unusual mobility may be congenital or acquired.

Congenital mobility is due to the presence of a *mesonephron*—that is, the kidney is partially or completely surrounded by peritoneum (like the colon), and is thus free to move about among the abdominal viscera. This condition is quite rare. It is sometimes distinguished as *floating* kidney.

Acquired mobility is much more common. It affects females more

often than males; and the right kidney is movable thirteen or fourteen times as often as the left. Sometimes both are affected at the same time. The age of the patient is mostly between twenty and fifty. It mainly results from conditions which stretch or relax the tissues and structures surrounding the kidney, especially the fatty capsule and the peritoneum. Perhaps the most frequent cause is repeated pregnancy, by which the peritoneum is dragged upon and stretched, and fails after delivery to recover its normal tension. But movable kidney is not confined to those who have borne children. Emaciation by reducing the fat surrounding the kidney may be a cause sometimes. Many patients have a pendulous abdomen, and general want of tone in the abdominal and pelvic tissues (*see* Glénard's Disease, p. 821). It is asserted by some that recurring congestion of the kidney, in association with menstruation, may lead to displacement. At any rate, an increase of size of the kidney from any cause must favour it. Tight-lacing has been charged with it, but it frequently occurs independently.

Symptoms.—The most common subjective symptom is a sensation of weight, or dragging, or pain in the loin or side of the abdomen affected; and this may be constant, aggravated by walking or exertion, and relieved by lying down. From time to time there may be severe attacks of so-called *strangulation* of the kidney (*Dietl's crises*), consisting of great pain and tenderness in the renal region, with scanty, high-coloured, and even bloody urine. There may be nausea or vomiting, and malaise; but generally not much pyrexia. Such an attack, which subsides in the course of a week or more, is probably due to twisting or kinking of the renal vessels by the movements of the kidney.

Rarely does a movable kidney produce any considerable pressure on surrounding organs, for its very mobility renders this unlikely; but œdema from pressure on the inferior vena cava has been recorded. Some gastric disturbances, such as nausea, flatulent distension, &c., are probably due rather to the nerve-connections of the kidney and of the stomach than to pressure on the duodenum, as suggested by some. Lastly, many patients suffering from displaced kidney are nervous, hysterical, neurasthenic, or hypochondriacal.

The evidence of movable kidney lies in its detection by palpation of the abdomen. In the majority of cases it is felt only in the flank of the side affected. Here a smooth, firm but not hard, rounded tumour may be felt, of the size of the kidney. If it lies between the last rib and the crest of the ilium, it can be pushed more or less in all directions, but most easily upwards towards the thorax, when it may get entirely out of reach, leaving the flank normal Often when the patient lies down, nothing is felt until she takes a deep breath, when the kidney glides down, and may be secured by dipping the fingers in above its upper extremity. If the organ be pressed firmly, or grasped, the patient experiences a sharp pain, or sickening sensation. The examination should be made

with both hands, one pressed firmly in between the last rib and the crest of the ilium, the other on the front of the abdomen. In some cases the kidney rises towards the front of the abdomen, or rests in the iliac fossa, or can be pushed over towards the middle line or beyond it. A further examination in the erect posture may help our estimate of the extent of the displacement. Apparently no difference in respect of mobility exists between the kidneys that possess a mesonephron and those that do not. It should be noted that a transitory albuminuria may result from too free manipulation.

Hydronephrosis from repeated kinking of the ureter is a possible result of this complaint; except for this, mobility of the kidney is rather a discomfort than a serious disease, and does not tend to a fatal result.

Diagnosis.—A movable kidney is likely to be confounded with small ovarian cysts, with distended gall-bladder, with tumours of the omentum, mesentery, stomach, pancreas, ascending or descending colon, with retained fæces, or with enlarged spleen. *Ovarian tumours* are movable only in directions determined by their pelvic connections, and cannot be pushed up into the loin. A *distended gall-bladder* has dulness continuous with that of the liver, but may be extremely movable. Its cystic nature may be recognisable. *Tumours of the alimentary canal* or *pancreas* have rarely a perfectly smooth surface, vary in shape and size, and are more continuously painful. An enlarged *spleen* is never behind the intestines; the lower it lies, the more it gets to the front of the abdomen, and it is always close under the parietes.

Treatment.—The object of treatment, if symptoms are severe enough to require it, is to retain the kidney in its normal position, and so prevent the weight, dragging, pain, and other sensations, as well as the strangulation which may result from kinking of the vessels of the ureter. Abstention from violent exercise may be enough in some cases, and rest in the recumbent posture will give temporary relief. But as getting about again brings on the troubles, it is desirable to attempt support of the kidney by some kind of pad, truss, or bandage. A spring truss, with a large pad pressing on the front of the affected loin, may be used; but is likely to lead to atrophy of the tissues upon which it presses. Better is a broad bandage, extending from the groin to the sixth or seventh rib, with a large pad sewn into it, in such a position as to press into the right flank and thus prevent the fall of the kidney. An air-pad, which can be inflated while in position, is often effectual. A further desideratum is to strengthen the abdominal muscles by suitable gymnastic exercises. The treatment of the symptoms of strangulation consists in complete rest, the use of poultices and hot fomentations to the loin and abdomen, and opium or morphia by injection or suppository. Severe cases of movable kidney justify the operation of *nephropexy*, by which an incision is made in the loin, and the kidney is stitched to the parietes. *Nephrectomy* may

be justifiable if nephropexy fails, and especially if the kidney is diseased as well as movable, and if the other kidney is sound.

RENAL CALCULUS

(*Nephrolithiasis*)

The following are the varieties of urinary calculi. The first five are the commoner forms; the others are much more rare:

1. *Uric Acid.*—Hard, round or oval in shape, smooth or finely tuberculated, sometimes faceted from contact; of yellowish, fawn, or reddish colour. They vary in size, from that of poppy-seeds to that of mustard-seeds or peas, and are occasionally very much larger. Frequently they exist in great numbers.

2. *Sodium Urate.*—Soft, not generally of large size.

3. *Calcium Oxalate*, or *Mulberry Calculus.*—These are very hard, rough or irregular on the surface, and of blackish-brown colour; when smaller they are smooth, rounded, gray or brown in colour. Generally they are solitary.

4. *Mixed Calcium and Ammonio-magnesian Phosphate*, or *Fusible Calculus.*—The mixed phosphates are precipitated in urine rendered alkaline by ammoniacal decomposition, such as occurs when the secretion is retained in the bladder or in a dilated pelvis. They rarely form the nucleus of a stone, but are deposited upon other calculi of uric acid or oxalate, upon foreign bodies (*e.g.*, in the bladder), and upon the inflamed mucous membrane of the bladder or of the renal pelvis. They may thus enormously increase the size of vesical stones, and in the pelvis may form concretions, which are moulded to all the infundibula and calices (*dendritic calculi*). The deposit is white, soft, and friable; and fuses under the blowpipe into a kind of enamel.

5. *Calcium Phosphate.*—White and chalky, rather smooth on the surface, with an earthy fracture, varying in size from that of a pea to that of a hen's egg.

6. *Calcium Carbonate.*—Small, very hard, smooth, gray, yellowish or bronze-coloured, and varying from minute grains up to stones the size of a hazel-nut.

7. *Cystine.*—Usually egg-shaped, the surface granular, glistening with crystals of yellow colour, looking translucent on section, with indications of a radiating structure, and rather soft in consistence. They become green on exposure. With a lens the hexagonal form of the crystals may be seen.

8. *Xanthine.*—In physical characters like uric acid calculi, but of a cinnamon colour, soluble in liquor ammoniæ and liquor potassæ. They are extremely rare, and have not been found in the renal pelvis.

9. *Urostealith.*—Soft, greasy concretions, which have been found in a few cases; one was shown to consist of about one-third cholesterin and fat, one-third uric acid, and some oxalates.

10. *Indigo.*—Once found in the renal pelvis by Ord as a calculus weighing 40 grains.

The majority of calculi consist of more than one of the above substances, and sometimes there are found alternating layers of uric acid, oxalate, and phosphates, laid one upon another under varying conditions of the urine. Phosphates and carbonates are deposited in alkaline urine; the remainder of the calculi above enumerated form in acid urine.

Urinary calculi vary much in size; they may be two or three inches in diameter, or they may consist of very small particles, and are then known as *gravel.*

The centre or nucleus (*i.e.*, the first-formed portion) of most calculi is uric acid; but within that, calcium oxalate or sodium urate has been found. Some calculi are deposited upon a nucleus of blood-clot, mucus, or renal casts, and the ova of Bilharzia hæmatobia may form the starting-point of renal stones. Most calculi are formed in the urinary tubules, and some even in the epithelial cells (Ralfe); and the frequency of uric acid and calcium oxalate as components of stone is, no doubt, determined by their relative insolubility. Another important factor seems to be the presence of some "colloid matrix," such as may be formed by mucus, blood, or perhaps the protoplasm of the epithelium-cell; since it has been shown by experiment that in the presence of viscid solutions a chemical precipitation does not take place rapidly in a crystalline form, but more slowly in the shape of granules, spheroids, and laminæ, which has been called *submorphous.* Such colloid matter, therefore, determines the form of the precipitated matter, and may also bring about the precipitation in a secretion overcharged with the relatively insoluble salt. According to Ralfe there is often an impairment of vital power in the renal cells, so that they fail to secrete uric acid or oxalate, and hence these substances are actually deposited in the cells, the cell substance acting as "colloid." Ultimately the calculus grows by accretion of other deposits upon it. Roberts showed that the precipitation of uric acid from the urine was favoured by great acidity of the urine, by excess of uric acid, by decrease of the urinary pigment, and by decrease in the urinary salines; and that it was retarded by the opposite conditions.

Ætiology.—Calculus is very much more frequent in the eastern part of England than in the middle or western parts; a difference which is not explained by the existence of a chalky soil in the former, since the majority of calculi from patients in these parts are of the uric acid variety. Stone is more common among the poor than the rich, more in males than in females, and perhaps more in early or late life than middle age. Ralfe pointed out that these were periods of vital impairment. For instance, febrile illnesses are common among children, and many suffer from malnutrition; on the other hand, in old age, besides the obvious waning of the powers generally, there is often local impairment

from urethral stricture, or prostatic enlargement, or diminished expulsive powers.

Symptoms.—(1) When the stone exists in the pelvis of the kidney, it may remain entirely latent, or it may give rise to lumbar pain, hæmaturia, albuminuria, or the passage of pus from the induction of pyelitis; and these changes may result from the presence of very small stones, or of the deposit known as gravel. (2) If it falls into the ureter, it may become impacted or move along with great difficulty, causing *renal colic*, hæmaturia, and, under certain circumstances, *obstructive suppression*. (3) The later effects of renal stones, either in the kidney, or after impaction in the ureter, are pyelitis of all degrees, pyelo-nephritis, perinephritis, perinephric abscess, hydronephrosis and pyonephrosis, the symptoms of which have already been described (*see* pp. 933, 935, 938).

Gravel and calculus are frequent causes of *lumbar pain*, which is often regarded as "lumbago" or muscular rheumatism. The lumbar pain or aching may be rendered worse by jolting or shaking. If the symptoms are of long duration, albumin, pus, or mucus may be passed, and from time to time blood in varying quantities. Occasionally, small calculi or gravel are discharged with the urine. The subsidence of symptoms entirely after a long period of activity may be due to the calculus becoming encysted.

Renal colic is caused by the spasmodic contraction of the muscular fibres of the ureter, irritated by the impaction or the passage of the calculus. It is comparable with biliary colic, and is characterised by intense pains, rigors, nausea, and vomiting. The pain is situated in the loin and flank of the same side, and radiates downwards and inwards to the groin and testicle; sometimes to the thighs, and even to the heel and sole of the foot; at others to the abdomen, chest and back. In the severer attacks the patient is doubled up with the pain, or writhes on the floor, and bursts out into profuse perspiration, or he becomes pale and collapsed, with quick, feeble pulse; but the temperature may be raised. With this there are nausea and vomiting, often a rigor, and sometimes even general convulsions. The testicle on the same side is retracted, and is swollen and very tender. The pain may be less for a time, but soon returns, and altogether it may last a few hours or a day or two, until at length the stone is passed into the bladder, or returns to the pelvis, when there is a sudden relief, and only an aching, smarting sensation in the side is left. The pain may, however, cease when the calculus still remains impacted in the ureter. During the attack, micturition is frequent and painful, and the urine is scanty, coming, perhaps, only by drops; and it may contain blood; and, indeed, a diagnosis of cystitis may be wrongly made. If the bladder be examined with the cystoscope, signs of irritation of the mucous membrane about the orifice of one or other ureter may be observed, and the urine flowing from either orifice may come too slowly, or may be turbid, or blood-stained. By examination of the abdomen the kidney may be found to be tender; or

the stone may be detected in the ureter, and its course watched from kidney to bladder.

Such an attack may occur spontaneously, or may be brought on by some movement which appears to dislodge the calculus from its position. Renal colic may recur in the same patient. This, of course, must depend on the number and size of the stones; obviously, if a stone gets back into the pelvis it may set up renal colic on again becoming impacted. Exceptionally a large number of stones in the pelvis of the kidney may be detected by a crackling sensation on palpation.

Obstructive suppression is distinguished by Roberts from the suppression which results from acute congestion or acute Bright's disease, and the symptoms in marked cases are strikingly different. It arises when both ureters are simultaneously compressed, as occurs in women when cancer of the pelvic organs invades the base of the bladder; or when one kidney has been disorganised, or excised, or otherwise placed *hors de combat*, and a calculus becomes impacted in the ureter of the healthy organ. This condition has been called *latent uræmia*. In some cases no urine is voided; in others, a certain amount may be passed in small quantities at long intervals, but it is clear, watery, of very low specific gravity—*e.g.*, 1006—and contains an extremely small quantity of urea or other solids: and there is no albumin, unless there is blood, or unless the urine is modified by the cystitis which accompanies cancer of the bladder. The patient's condition is not at first materially altered. He may eat as usual, but he loses muscular power, and becomes sleepless, and after some five or six days he is seized with muscular twitchings or jerkings, affecting the arms, legs, and trunk. The pupils are contracted, the temperature of the body falls, the breathing is slow, panting, and laborious, the mouth and tongue are dry, and there is great thirst and there may be troublesome vomiting. The muscular twitchings continue, and the patient becomes restless, indifferent, and drowsy, but neither convulsion nor coma occurs. Death, as a rule, ensues from nine to eleven days after the commencement of the obstruction, and is very rarely postponed beyond this. Recovery may take place if the obstruction is removed by the passage of the calculus or by the breaking down of any new growth. Roberts points out that there may be obstructive suppression of a less dangerous kind in the course of a double hydronephrosis; and Bradford shows that symptoms identical with the above may arise when the functions of the kidney, hitherto healthy, are disturbed by distant lesions, presumably acting reflexly, or by toxic influence.

Diagnosis.—The typical symptoms, lumbar pain, hæmaturia, and albuminuria—may be caused not only by a medium-sized calculus, but also by fine gravel and uric acid crystals, which will readily pass the ureter; and in these cases testicular pain and frequency of micturition may also be present. The diagnosis may be made by rendering the urine alkaline, when the symptoms will quickly diminish or cease altogether. A severe attack of colic, associated

with hæmaturia and testicular pain and retraction, is very strong evidence of calculus; but other foreign bodies besides calculus may set up colic while passing the ureter—*e.g.*, masses of viscid mucus the result of pyelitis, clots of blood which have formed in the renal pelvis, fragments of tissue in cancer of the kidney, and, exceptionally, hydatid daughter-cysts.

The influence of the seat of the hæmorrhage upon the character of the urine has been mentioned (*see* p. 905).

Any disease leading to hæmorrhage from the kidneys may for a time be mistaken for calculus, since hæmaturia may occur without other symptoms being prominent. In *cancer* of the kidney hæmorrhage is often more abundant, and more continuous. Gelatinous red lumps appear in the urine after the blood; and sometimes cancer-cells may be found by the microscope. The presence in due time of a tumour will help the diagnosis. Calculus is more likely in a young patient, but either may be present in middle or old age. *Tubercle* of the kidney may closely simulate calculus, by lumbar pain, frequent micturition, pus in the urine, and even blood. In the former there may be a family or personal history, or present indications of tubercle; hæmaturia and renal pain are less prominent and characteristic. *Cystitis* is simulated by the frequency of micturition which occurs in renal calculus, especially if hæmaturia is absent. Previous attacks of lumbar pain, and the acid reaction of the urine, if pus is present, are in favour of renal origin. *Hysteria* may simulate renal pain, and requires very careful consideration.

In difficult cases diagnosis may require manual examination of the kidney in the loin, the use of the cystoscope, and the Röntgen rays, which will often show the presence of a calculus. A metallic sound may be passed into one or other ureter, so as to detect the stone by contact. Lastly, an exploratory incision in the loin over the kidney is justified in some cases.

Some differences have been noted in the symptoms dependent on the chemical nature of the calculus. *Uric acid* calculi produce the least pain, which is dull and oppressive, with a sense of weight. The urine is acid; bleeding is frequent, not excessive, and periodic, apart from exertion. The mucous deposit is yellowish, or rusty. *Calcium oxalate* causes acute pain referred to a particular spot, as well as shooting pain in the ureter, shoulder, or epigastrium. The urine is less acid, bleeding is abundant, and the mucus is glairy and greenish. *Phosphates* cause severe unremitting pain, attended with exacerbations. The urine has been at some time alkaline, there is much muco-pus or flocculent mucus, which may be tinged with blood, though free bleeding is rare.

Treatment.—The frequent occurrence of gravel, which is nearly always uric acid, should be met by the administration of drugs to render the urine less acid, or even alkaline. The most efficient are the citrate, acetate, and bicarbonate of potassium, which may be given in 30 or 40 grain doses in 3 or 4 ounces of water two or three times daily, or in one larger dose before the night's rest, during

which time the tendency to uric acid precipitation is greatest (Roberts). Pure or distilled water, or Contrexéville water, may be also drunk, and the diet should consist largely of farinaceous and vegetable food. On the other hand, alcoholic liquors and highly nitrogenous food should be avoided. When, in addition, lumbar pain, and a trace of albumin suggest that the precipitation is taking place in the kidney, the same line of treatment may be pursued, and so long as the deposit is only minute it may be washed away by the urine, and the alkalisation of the urine will prevent any more being formed. Ralfe recommended turpentine in capsules every morning or twice daily. Benefit is often derived from residence at Vichy, Vittel, Contrexéville, Ems, Carlsbad, Salzbrunn, Tarasp, Neuenahr, or Wildungen, where the waters are alkaline or saline. As a preventive of stone exercise is desirable; but the encystment of a large calculus is likely to be favoured by rest in the recumbent position, inclining to the affected side.

If a calculus has actually formed and is stationary in the pelvis of the kidney, it is doubtful if it can be dissolved by any medical treatment. When therefore the symptoms are very severe and remain unrelieved by treatment or if the patient is prevented from following his livelihood, *nephrotomy* or *nephrolithotomy* should be performed; or *nephrectomy* if the kidney is hopelessly damaged.

Treatment of Renal Colic.—Anodynes are here required, both to relieve the intense pain, and because they may also relax the spasm of the ureter, and so facilitate the escape of the stone. If the pain is severe, a morphia injection should be at once given; or morphia or opium may be given internally, or in suppositories, or chloroform or ether may be inhaled. Locally, hot poultices, hot fomentations, belladonna applications, or the hot bath should be used. The patient should be at rest, and warm diluent drinks, barley-water, &c., should be taken from time to time.

MORBID CONDITIONS OF THE URINE INDEPENDENT OF PRIMARY DISEASE OF THE URINARY ORGANS

DIABETES INSIPIDUS

It has been already shown that polyuria, or excessive secretion of urine, may arise from various morbid conditions, such as chronic granular kidney, lardaceous disease of the kidney, and hysteria. But there are two forms of polyuria, which have long been known under the equivalent term diabetes (διαβαίνω = I go through), and which require special description. In the more common form, *diabetes mellitus*, sugar is present in the urine at the same time; in the other form there is no sugar, and it is called *diabetes insipidus*.

Ætiology.—Diabetes insipidus is a comparatively rare complaint. It occurs mostly in early adult and middle age, but sometimes in quite young children, and it is more frequent in males than in females. It cannot always be traced to a definite cause, but it has occasionally followed upon blows or injuries to the head, emotional disturbance, or convalescence from acute diseases. Family predisposition has also been recorded, the disease being handed down from parents to children. A nervous origin is suggested by the above, and this is supported by the knowledge of the experiment of Bernard, who produced polyuria by puncturing the fourth ventricle at a point a little above the centre for the production of sugar in the urine. Tumours of the brain and cerebral lesions due to syphilis are occasionally accompanied by marked polyuria and polydipsia in addition to the purely nervous symptoms.

Symptoms.—These begin either insidiously or suddenly; sometimes they have followed immediately upon the ingestion of a large quantity of water. The prominent symptoms are the enormous quantity of water passed, and the great thirst by which the patient is led to replace the loss. The urine may reach fifteen, twenty, or even forty pints in the twenty-four hours. It is very pale, almost like water, of specific gravity 1002 to 1005, and faintly acid in reaction. The percentage of solid constituents is, of course, small; the daily excretion of urea is generally normal, but may be much increased if the appetite is excessive. Sometimes *inosite*, or muscle-sugar, has been found; but it is also present in some cases of diabetes mellitus and Bright's disease, and even in health after large quantities of water have been drunk. Quite exceptionally, a trace of albumin is present, and more often minute traces of grape-sugar. The thirst is excessive and uncontrollable,

the patient being obliged to drink large quantities of water; and in most cases the amount ingested is in excess of the urine passed.

Other symptoms are the following: The mouth and tongue are usually dry, the skin is dry, and the temperature is normal. The appetite is often poor, sometimes unaffected; exceptionally, however, it is enormous, as it is so often in diabetes mellitus. The bowels are regular, or only slightly constipated. Beyond this, the patient may be in the enjoyment of very good health, and he finds the diabetes an annoyance rather than an illness. But often, especially in the severe cases, there are emaciation, weakness, and languor; the sleep is much disturbed, and there is mental depression or irritability of temper; occasionally the sexual powers are abolished.

The course of the disease is variable. If it arises from injuries to the head, it may be of short duration; when it is due to definite cerebral lesions, its course will be determined by them. Spontaneous and idiopathic cases may last for years, and are mostly intractable. They are rarely fatal, except from the intervention of other illnesses, especially phthisis and pneumonia; occasionally glycosuria has supervened, and the case has become one of diabetes mellitus.

Morbid Anatomy.—The lesions which are at all constantly found in diabetes insipidus are very few. Dilatation and hypertrophy of the bladder, dilatation of the ureters, and enlargement of the kidneys may be seen, and are attributable to the prolonged pressure of large quantities of urine. In addition there may be found in a few cases the cerebral lesion which has been recognised as the cause, or the disease of the lung which has been fatal.

The polyuria is probably due to dilatation of the renal vessels from loss of control by their vasomotor nerves. It clearly has a nervous origin.

Diagnosis.—The enormous quantity of pale urine, of low specific gravity, without abnormal ingredient, and the accompanying thirst, are distinctive. But care must be taken to exclude other forms of polyuria, such as those from *Bright's disease* and *hysteria*. In the former there is generally at some time or other a distinct trace of albumin, the quantity of urine is not so considerable, and other indications are present, such as high arterial tension and cardiac hypertrophy. In hysteria the condition is but temporary. The urine of *diabetes mellitus* is at once distinguished by its higher specific gravity and by the presence of glucose.

Treatment.—The removal of the cause is rarely possible, and the treatment generally resolves itself into attempting to influence the nervous system by certain drugs. Valerian often has a very good effect. It should be given at first in 5-grain doses of the powdered root three times a day; the dose should be increased by 5 grains at a time, and, if necessary, to the extent of 2 or 3 drachms in the day. The infusion or extract of valerian and valerianate of

zinc (up to 20 grains daily, in divided doses) may be substituted. Ergot, ergotine, suprarenal extract, codeine, bromide of arsenic, bromide of potassium, carbolic acid, nitro-glycerine, sodium salicylate, daily injections of strychnine ($\frac{1}{30}$ to $\frac{1}{60}$ grain) have also been used; and good results have been recorded with antipyrin in 8-grain doses every two or three hours, so as to reach 60 or 70 grains in the day. In syphilitic cases full doses of potassium iodide, with or without mercury perchloride, should be given. The constant galvanic current may be applied over the medulla oblongata or upper part of the spinal cord; or one pole may be applied to the loin, and the other to the hypochondrium on the one side for a few minutes, and the same on the opposite side; or the anode may be applied to the nape of the neck, and the kathode first to the loins and then to the epigastrium (Külz).

DIABETES MELLITUS

The characteristic feature of this illness is the passage of large quantities of urine containing glucose or dextrose. The term *glycosuria* means the presence of sugar in the urine: if it is temporary and not accompanied by *polyuria* it is not technically diabetes. But a persistent glycosuria may be a serious condition, and may subsequently develop into pronounced diabetes.

Ætiology.—It occurs twice as frequently in males as in females. It is rare in childhood, but occurs at puberty, and most often in middle life and advancing age. It is more frequent in fair than in dark people; and it appears to be more prevalent in urban and manufacturing than in rural districts (Roberts), and in the eastern than in the western counties of England. Considerable differences are also noted in its prevalence in various parts of the world; in Europe it is frequent among the Jews. It appears to be hereditarily transmitted; it may affect two or more brothers and sisters, or a man and his wife; and it has sometimes occurred in two or three children of one family at, or soon after, birth. It often appears to be caused by some disturbance of the nervous system, such as overwork, anxiety, or emotion, and occasionally follows an injury to the head. There is a possible association with toxic conditions, such as the infections of malaria, influenza, scarlatina, diphtheria, tonsillitis, and the less definite infections, gout and rheumatism. Undue indulgence in starchy and saccharine food, or in alcohol, is sometimes an antecedent. Of definite lesions in the body previous to the occurrence of diabetes may be mentioned exophthalmic goître, and acute and chronic changes in the pancreas, which may be found after death; but there is an increasing number of cases in which subacute or chronic lesions of this organ without glycosuria have been followed by true diabetes mellitus.

Diabetes has also sometimes supervened upon lesions of the liver, suprarenal glands, and intestine.

The Condition of the Urine.—The actual quantity of sugar in the urine in cases of diabetes varies from a mere trace up to a maximum of forty grains to the ounce. From eight to ten grains per ounce is the more usual amount. The daily excretion is often 6000 to 7000 grains The presence of sugar is commonly accompanied by other changes in the urine. Its quantity is increased to ten, fifteen, or twenty pints per diem, and the specific gravity is raised by the presence of so much sugar to 1035, 1040, or 1045. It is said to have been even 1060 or 1070, but this must be rare. The colour of the urine is generally pale yellow, or almost like water; it has a sweetish odour like hay, and a sweet taste. The reaction is acid. If it contains much sugar it will leave a crystalline deposit as it dries upon linen or elsewhere. The urea of diabetic urine is in excess of the normal, sometimes very much so; uric acid is either unaffected, or, according to some, is below the normal. Phosphates and sulphates are usually in proportion to the urea.

In the higher degrees of diabetes, the urine will probably contain the substances known as the *acetone* bodies. These are β-oxybutyric acid, diacetic (aceto-acetic) acid, and acetone. The last may be in the urine alone, and indeed there may be a slight amount even in normal urine; but if diacetic acid is present acetone will accompany it, and if oxybutyric acid is present, the other two compounds will be there also. Oxidation converts oxybutyric acid into diacetic acid, and this breaks up with further oxidation into carbon dioxide and acetone. When the urine contains oxybutyric acid, ammonia is always present in large amount.

Clinical Tests for Sugar.—The particular form of sugar contained in diabetic urine is grape-sugar, or glucose ($C_6H_{12}O_6$), and this has the property, when heated with a salt of cupric oxide, of reducing it to cuprous oxide or suboxide. This test, in one or other form, is the one most commonly employed.

Fehling's Test.—This is a more convenient way of applying the method first adopted by Trommer, in which a few drops of a solution of cupric sulphate are added to urine in a test-tube, and then about half a drachm of liquor potassæ, when, if sugar is present, the cupric oxide at first precipitated by the potash is redissolved. On applying heat to the solution, a thick precipitate of cuprous oxide at first yellow, quickly changing to orange and red-brown, is thrown down.

Fehling's solution consists of equal parts of (1) a cupric sulphate solution of 34·63 grammes in half a litre of distilled water, and (2) caustic potash, 100 grammes, and sodium potassium tartrate 173 grammes in half a litre of water. The two solutions are mixed. After some weeks decomposition takes place, and renders the test uncertain. This may be prevented by keeping the two solutions separate, and only mixing them for use, or at short intervals; or the altered mixture may be again rendered fit by the addition of a piece of caustic potash.

In Pavy's modification of Fehling's test, the proportions are as follows: Crystallised copper sulphate, 34·65 grammes; caustic

potash, 170 grammes; potassium sodium tartrate, 170 grammes; distilled water to 1 litre. Pavy prefers the larger amount of alkali, because it prevents precipitation by the small quantity (average ·279 per cent.) of reducing substance present in normal urine.

When half a drachm of one of these reagents is boiled with an equal quantity of saccharine urine the cuprous oxide is thrown down as a yellow precipitate, changing to orange, and red-brown. If the quantity of glucose is small, the precipitate is at first bluish-green, and then becomes yellow; or if smaller still, the precipitate remains bluish-green from the presence of unreduced cupric salt, or discoloration takes place without precipitate. The relative proportions of the urine and the test-fluid are of importance. When the former has a sp. gr. of not more than 1020, the quantities should be equal. If the sp. gr. is higher, the urine should be diluted to 1020 or less, and an equal quantity of the Fehling's test used; or with the increasing sp. gr. larger proportions of the test should be employed. Thus to 2 c.c. of urine at sp. gr. 1025, 2·5 c.c. of the test; at sp. gr. 1030, 3 c.c. of test; at sp. gr. 1035, 3·5 c.c., and at sp. gr. 1040, 4 c.c. of test (Kellas and Wethered).

Uric acid and creatinine also reduce cupric oxide, but together they only form one-fourth of the reducing agents normally present in the urine. Creatinine in excess delays the precipitation of the cuprous oxide, by the evolution of ammonia during boiling. Pavy suggests a test-meal as a means of determining the significance of a doubtful reaction; if no effect is produced upon the urine in two or three hours by a plentiful allowance of starchy and saccharine food, it may be concluded that nothing abnormal exists.

Moore's Test.—Half a drachm of saccharine urine, heated in a test-tube with an equal quantity of liquor potassæ, changes to a rich red-brown. The test is uncertain for small quantities, and is rarely used.

Phenyl-hydrazine Test.—A test-tube is filled for about half an inch with phenyl-hydrazine hydrochlorate, and for another half-inch with sodium acetate. The test-tube is then half filled with urine, and the whole is boiled over a spirit-lamp and kept boiling for two minutes. The yellow sediment is examined after about half an hour, when it will show, under the microscope, sheaf-like clusters of fine crystalline needles (phenyl-glucosazone), which melt at 205° C.

With small quantities of glucose the urine and test-substances should be heated for 30 to 60 minutes in a water-bath. If the deposit is amorphous, dissolve it in hot alcohol, dilute with water, and boil to expel the water, when the osazone will separate in crystals.

Safranine Test.—Crismer's safranine test is strongly recommended by some (Allen, Kellas and Wethered). If 2 c.c. each of a solution of safranine (1 in 1000), of caustic potash (B.P.) or caustic soda (4 per cent.) and of saccharine urine are boiled together, the bright red colour changes to clear yellow, and the solution becomes turbid from the deposition of a white substance. The reaction occurs with a sugar-content of ·1 per cent.; it is not affected by creatinine,

creatine, mucin, uric acid, or urates, and only very slowly by albumin. If the red colour is discharged—and it may be so by the small amount of sugar contained in healthy urine—successive quantities of 2 c.c. of safranine solution should be added, and the mixture again boiled after each addition, until ultimately the red colour persists. If more than four or five measures are required the urine is distinctly diabetic.

Fermentation Test.—If a small quantity of yeast (washed free from any starch or sugar) be added to the urine, and this be set aside in a warm place for some hours, the glucose will be converted by fermentation into alcohol and carbonic acid. If now the specific gravity be taken, and compared with what it was before the experiment, or better with that of a duplicate specimen, placed under similar conditions except for the presence of yeast, it will be found that there is a loss of density corresponding to the destruction of the glucose. If the test-tube be filled entirely and inverted in a saucer, the carbonic acid gas, as it forms, will collect in the upper part and displace the urine.

Polariscope.—By circular polarisation not only the presence but the quantity of sugar can be accurately estimated. But the instrument is not generally available for clinical use. The glucose found in the urine rotates the polarised ray to the right, and is hence called *dextrose.*

Fallacies.—With the copper test a small amount of reduction may take place from the presence of glycuronic acid, uric acid, hippuric acid, and urates when no sugar is present. Discoloration after prolonged boiling should be distrusted. *Glycuronic acid* ($C_6H_{10}O_7$) reacts to Fehling's test, and is also dextro-rotatory. It is thus readily mistaken for glucose, but it does not answer to the fermentation test; and it gives with the phenyl-hydrazine test a brownish amorphous deposit, which melts between 115° C. and 150° C. The supposed occurrence of glycosuria after the administration of morphine, curare, and chloroform vapour is due to this substance; chloral, butyl-chloral, camphor, copaiba, cubebs, salicylic acid, and tannic acid also produce it. In nursing women *lactose* may pass into the urine, and give some reactions of dextrose; with the phenyl-hydrazine test it gives crystalline needles in spherical clumps, which melt at 200° C. *Lævulose,* or fruit-sugar, which rotates the polarised ray to the left, and *pentose* ($C_5H_{10}O_5$) are found on rare occasions; the latter precipitates with Fehling's solution and phenyl-hydrazine, but does not affect polarised light, and does not ferment.

Creatine and mucin, as well as creatinine, delay the precipitation of cuprous oxide when only very small quantities of sugar are present.

Quantitative Estimation of Sugar.—In any cases of diabetes undergoing treatment, it is desirable to estimate from time to time, if not daily, the amount of sugar passed. For this purpose all the urine passed in twenty-four hours must be collected, and a specimen of the mixed urines may then be submitted to analysis by one of the following methods.

The Copper Test.—This can be employed quantitatively, by heating a measured amount of Fehling's or Pavy's test-fluid in a capsule, dropping the urine into it from a graduated pipette, so as to note the exact quantity of urine required to reduce the cupric oxide, and thus discharge all the blue colour.

By means of Pavy's *ammoniated cupric solution*, the precipitation of the suboxide is prevented, and the exact steps of the decoloration of the copper solution can be more accurately watched. The solution consists of—

Cupric sulphate	4·158 grammes	or	36½ grains.
Potassium sodium tartrate	20·400 „		178 „
Caustic potash	20·400 „		178 „
Strong ammonia (sp. gr. ·880)	300 c.c.		6 fluid oz.
Water	to 1 litre		to 1 pint.

The caustic potash and the tartrate are dissolved in some of the water, and the copper salt in another portion; these are then mixed, and when cold, the ammonia is added, and the whole is diluted to the specified bulk. Ten cubic centimetres of this solution are exactly decolorised by ·005 gramme of the sugar. As in the preceding method, a burette is used, into which the urine previously diluted to the extent of 1 in 20, or even 1 in 40, should be placed; the nozzle of the burette passes through a cork in a small flask containing the 10 c.c. of the copper solution, diluted with 20 c.c. of water. Another glass tube in the cork allows the escape of steam. Heat is then applied to the flask, and the urine is gradually dropped in from the burette. When the colour is all discharged, the result is read off and the desired calculation made. Pavy recommends, for clinical convenience, the use of hermetically-sealed glass tubes, each containing 10 c.c. of the ammoniated cupric solution.

Safranine Test.—A rough estimation can be made with this by proceeding as indicated above (*see* p. 967). Each 2 c.c. corresponds to ·1 per cent. of glucose or equivalent substance. If there is much sugar the urine must be diluted proportionately.

Fermentation.—This can be used for quantitative purposes by taking the specific gravity before and after fermentation for twenty-four hours: every degree of specific gravity lost corresponds to one grain of sugar per ounce. Thus, if the specific gravity is reduced from 1040 to 1025, there are 15 grains of sugar per ounce. Or the gas may be collected in a graduated tube from a measured quantity of urine, as in the convenient apparatus of Gans. The graduations give the percentage of sugar.

Clinical Tests for the Acetone Series.—The test for *diacetic acid* is a solution of perchloride of iron, which first precipitates the phosphates, and then, as they are dissolved by a further addition of the solution, a claret-red colour is produced. *Acetone* is detected by dropping a small piece of sodium nitro-prusside into a few c.c. of urine, and then a little caustic soda. A cherry-red colour is developed which soon fades; an excess of acetic acid now added

produces a carmine-red colour. Or strong ammonia solution is carefully floated on the top of a solution of sodium nitroprusside and urine: in from one to three minutes a ring of magenta colour appears at the junction of the two fluids, and spreads upwards into the ammonia (Jackson-Taylor). *Oxybutyric acid* can be estimated by the use of the polarimeter. It is lævo-rotatory, and a marked difference between the results given in the estimation of dextrose by Fehling's solution and by the polarimeter is attributable to, and may be estimated as, oxybutyric acid.

Symptoms and Course.—The onset of diabetes is sometimes insidious; the patient only gradually notices that he drinks more fluids and passes more urine than normal; or he may complain of debility and loss of flesh rather than any alteration in his urine. In other cases the symptoms develop almost suddenly after chill, after quenching thirst with large draughts of water, after severe emotional disturbance, or after injury.

The characteristic symptoms soon become unmistakable—namely, frequent and abundant micturition, great thirst, generally a very large appetite, physical weakness, and loss of flesh. The appetite is sometimes enormous, but in other cases it is but little affected, and often fails towards the end. The mouth and lips are dry, the tongue large, red, raw, and "beefy"; and there is generally a sweet taste in the mouth. The digestion is, as a rule, good, and patients may have no difficulty in disposing of the large quantities of animal food they take. The bowels are generally confined. The skin is harsh and dry, and the temperature often slightly below the normal. At the same time, nutrition is profoundly affected; the patient rapidly loses flesh, and becomes excessively weak; he is indisposed to make any mental effort, and is depressed and irritable. The teeth are carious, or become loose and fall out. There is often loss of virility in men, and in women the menses may cease.

The progress of the disease is very variable: some cases begin suddenly, and end in death in the course of from two to five weeks from the commencement; others may last, more or less influenced by treatment, for two, three, or four years; in other cases, again, the disease can be held in check for long periods by suitable treatment, and life may be prolonged for several years. An important point of distinction between the first and last class of cases is that the former are progressive from the first, and are little if at all influenced by diet, since sugar continues to be excreted, even though the carbohydrates are withdrawn entirely from the food; whereas the latter are very favourably influenced by depriving them entirely of saccharine and starchy foods, so that not only does the sugar disappear from the urine, but the urine decreases in quantity, and all the troublesome symptoms are arrested for a time. A relapse, however, is tolerably certain to take place at some future date, to be again, or many times, controlled by diet. Eventually this fails, and phthisis, coma, or some other complication carries off the patient. The most rapid and intractable cases occur in young persons, while

it is especially middle-aged and elderly patients in whom the disease is easily controlled by diet and treatment.

Complications.—In the course of diabetes a number of complications are liable to occur. The irritation of the saccharine urine may excite in women a troublesome *pruritus pudendi*, and in men, *balanitis*. The skin, besides being harsh and dry, is not infrequently the seat of *eczematous* or *lichenous* eruptions, generally dry in character. *Carbuncles* and *boils* are especially liable to occur in various parts of the body, and the former are not infrequently the cause of death. A form of *xanthoma* has also been seen in diabetes. There is sometimes *gangrene* of the toes or of an entire limb; but this is associated either with atheromatous arteries or with peripheral neuritis. The heart is not generally affected in a marked manner, but it may become weakened, and the pulse may be slow, or quick and irregular; occasionally, also, œdema of the feet, not due to nephritis, may indicate the same. Some of the most serious complications are those arising in the lungs, which become the subject of *pneumonia* or *tubercular phthisis*. The pneumonia is of the lobar variety, and sometimes results in gangrene of the lung. The phthisis is usually very rapid, bringing about death in from two to five months from its first appearance.

Vision is affected in diabetes in several ways. The most important is the formation of *cataract*, which is always symmetrical, and develops rapidly in the young and middle-aged, more slowly in old people. Other changes are: *Defects of accommodation*, from paralysis of the ciliary muscle, sometimes coming on quite suddenly; *retinitis*, somewhat resembling that of albuminuria, with white spots and hæmorrhages; and *atrophy* of the optic nerve. *Amblyopia*, without visible ocular changes, like that of uræmia, also occurs.

Diabetic Coma.—This name has been given to a group of symptoms, which are not infrequently the final cause of death in diabetes; they suggest an origin in the nervous system, but are undoubtedly due to a toxæmia, probably by excess of acids (*acidosis*). The onset is often gradual and insidious, but may be indicated by loss of appetite, by a rapid fall in the quantity of urine and of sugar passed in the day, and by obstinate constipation. Sometimes there is severe abdominal pain. The patient then rather rapidly falls into a condition of collapse rather than coma. The pulse is quick and feeble, the surface cold, the features pinched, and the extremities livid. He lies with the eyes half open, taking no notice of his surroundings; and though he can be roused by a question, he answers, if at all, in a dazed manner, as if only half comprehending it. The breathing in these cases is peculiar; it is slow, deep, and sighing in character; the movements of the chest are very extensive, but without the rapidity of ordinary panting from exertion. At the same time, examination of the chest reveals nothing abnormal. This form of breathing has been called *air-hunger*, a name which does not distinguish it from other forms of dyspnœa. In many cases a sweetish, fragrant, or ethereal odour, likened to the

smell of apples by some, may be noticed about the bed of the patient; it has been attributed to acetone. This condition may last from one to three days, when the pulse gets more and more feeble, though the heart may be beating forcibly, the patient more apathetic, and finally quite comatose; and death, with rare exceptions, ends the scene. Occasionally there is a little muttering delirium, but rarely convulsions. In some cases the symptoms are much more rapid; without any warning, often after some excitement or unusual fatigue, the patient becomes collapsed, with a quick, feeble pulse and livid extremities, and dies after twenty-four or thirty-six hours. It seems probable that there is no essential difference between these extremely rapid cases and the slower cases first mentioned, in which the peculiar character of the respiratory movements is such a prominent feature.

Coma may occur in almost any case of diabetes: acute cases in young people occasionally terminate thus in the course of a few weeks or months; or more chronic cases, apparently going on well, may be suddenly attacked. The rapid change from an ordinary to a diabetic diet has seemed sometimes to be the exciting cause.

Among other less important nervous symptoms in the course of diabetes may be mentioned—cramps; neuralgia, especially sciatic, occipital, and trigeminal; hyperæsthesia, anæsthesia, absence of knee-jerk, extensor paralysis with high-stepping gait or definite ataxia, and other symptoms of *peripheral neuritis*. Cerebral hæmorrhage may accidentally complicate diabetes and produce coma, which must not be confounded with that due to diabetic poisoning; the presence of paralysis would distinguish the former.

A trace of albumin is occasionally found in the urine, and this is sometimes due to a tubal or interstitial nephritis.

Fatal Termination.—Over 50 per cent. of cases of pronounced diabetes die of coma, another 25 per cent. die of phthisis or pneumonia, and the remainder of Bright's disease, cerebral hæmorrhage, gangrene, carbuncle, or other complications. Among the cases dying of coma, a certain proportion are also affected with pneumonia or phthisis, from which the patients would, no doubt, have died, had they not been carried off by the more rapid nervous symptoms.

Morbid Anatomy.—In a certain proportion of cases, the *pancreas* is diseased. Most frequently it is the subject of atrophy, or fibrosis, or the two combined; and other changes found in these circumstances are fatty transformation of large parts of the gland, suppuration, hæmorrhage, cancer, calculus in the ducts, and cysts. In many cases, especially those of short duration, the *post-mortem* appearances of other organs differ very little, if at all, from the normal: in older cases the pathological lesions due to the complications are found. There is often enlargement, with some softening and congestion of the *kidneys*, which otherwise may look perfectly normal, or may present some fatty change. Various alterations of the tubal epithelium have been noted under the microscope: A

vesicular or swollen and translucent condition of the cells of the connecting tubes by Cantani and S. Mackenzie; glycogenic degeneration of the epithelium of Henle's tubes by Ehrlich; and necrosis of the epithelium by others. The *bladder* is often hypertrophied. The *liver* presents nothing abnormal to the naked eye. It is stated to contain less glycogen than a healthy liver. The *lungs*, in cases dying of coma, are generally congested and œdematous, but otherwise healthy. In other cases croupous forms of pneumonia or the acute caseating phthisis already mentioned are found. The *heart* may present some atrophy of its muscular fibres. The *blood* is, in some cases, quite natural in appearance, in others it is black and tarry; in others, again, it presents a peculiar pink or strawberry colour, and on being placed aside a creamy layer collects on the surface. In this fat has been demonstrated, and the condition has been called *lipæmia;* but, on the other hand, the granules constituting the creamy layer certainly sometimes differ from those of true fat, and are partly proteid in nature. In some of these cases fat-embolism of the pulmonary capillaries occurs; and the condition of the blood has been recognised during life in the retinal vessels. Diabetic blood contains from two to four times the amount of sugar found in health; and oxybutyric acid has been discovered in it. The brain, medulla oblongata, spinal cord, and sympathetic system have been often examined for lesions which would explain the symptoms; but the wide perivascular spaces, thick arteries, pigment deposits outside the arteries, &c., which have been described, are not peculiar to, or constant in, diabetes. Peripheral neuritis occurs, but is no doubt secondary. It is with extreme rarity that any coarse lesion, such as a tumour involving the glycosuric centre in the medulla, is found as the cause of a typical case of diabetes.

Pathology.—The commonly adopted explanation of the presence of sugar in the urine is as follows: It is believed that in health sugar, whether taken directly, or formed from starch in the alimentary canal, is arrested in the liver, by being converted into a less diffusible substance, glycogen, which is deposited in the hepatic cells; that this glycogen is gradually again converted, by the action of a ferment, into glucose; and that glucose is decomposed and destroyed in the blood by the processes of respiration and muscular contraction as rapidly as it is formed, so that it never appears in any appreciable quantity amongst the urinary excreta, though it is found in the blood to the extent of ·5 per 1000. Thus glycosuria might arise from defective relations between the liver and the ingested carbohydrates, the liver failing in function, or the carbohydrates being in excess: or, secondly, from defective relations between the sugar supplied from the liver, and the processes (respiratory or muscular) which destroy it, the sugar being in excess, or the destructive processes failing.

Pavy holds that if sugar gets into the blood, as assumed in the above theory of the processes in health, it would appear in the urine much more abundantly than is actually the case. He states

that in health the carbohydrates of the food are (1) assimilated by synthesis into proteid by the lymphocytes of the villi; (2) converted into fat by the agency of the epithelial cells of the villi; and (3) whatever escapes the villi is converted into glycogen in the liver. Thus sugar in any quantity neither reaches the general circulation, nor is subsequently got rid of by the respiratory or other process; although a minute amount is present in normal urine and is proportionate to the quantity normally in the blood. If these assimilative processes fail, sugar is not converted into proteid, fat and glycogen, but enters the blood and escapes by the urine, causing glycosuria. Quantities of all kinds of sugar can be given which will be in excess of the normal powers of assimilation, and glycosuria will result. But the condition is transitory, dependent on external causes, and is not diabetes.

But in the worst cases of diabetes the sugar in the urine is derived not only from the food carbohydrates, but also from the food-proteids, and possibly from the tissue-proteids, since sugar is passed even when no carbohydrate is ingested. When this destructive metabolism of the proteids takes place, not only sugar appears in the urine, but also the compounds of the acetone series, to which reference has been made; and their presence in the blood at the same time tends ultimately to the production of coma.

To this form of glycosuria Pavy has given the name *composite diabetes*, to distinguish it from the less serious and more manageable condition, in which sugar alone is found in the urine; and the latter he calls *alimentary diabetes*. Undoubtedly the one may pass into the other.

Various explanations have been given of the way in which the nervous system influences the hepatic functions in diabetes: By vasomotor paralysis in the liver (Pavy); by general high blood-pressure causing dilatation of the hepatic vessels (Brunton); by the influence of the nervous system upon chemical processes in muscle (Bunge); by special influence upon the formation of ferments.

But the relation of the pancreas to diabetes has also to be taken into account. Glycosuria has been produced by the complete extirpation of this gland in animals, but fails to occur if only a small portion of the gland is left *in situ*; and we have seen that in quickly fatal cases the pancreas is often diseased. The theory adopted to explain this association is that the pancreas, besides pouring the pancreatic juice into the duodenum, has an "internal secretion," which favours the splitting up or destruction of dextrose, and that in the absence of this secretion the dextrose persists and appears in the urine. Pavy supposes that the pancreas interferes by enzyme action with the assimilation processes in the bowel and liver; and to a similar enzyme action he attributes the breaking down of the proteids in the severer, composite cases.

However this may be, it has been shown by Bayliss and Starling that the external secretion of the pancreas is stimulated by a substance called *secretin*, produced by the duodenal mucous membrane;

and it has been suggested that some cases of diabetes may be due to a failure on the part of the duodenum in regard to this function.

The temporary appearance of sugar in the urine, which can only be called *glycosuria*, and is not included under either alimentary or composite diabetes, may be observed under the following conditions: after the ingestion of very large quantities of sugar; after attacks of whooping cough or asthma; in the convulsions of epilepsy or in the coma or convulsions of apoplexy; as a result of some other coarse cerebral lesions. It may result also from the action of carbonic oxide, phloridzin, and preparations of the thyroid and suprarenal glands. Phloridzin-glycosuria appears to be due to the action of the renal cells in separating dextrose from some substance brought to them by the blood. The influence of the thyroid gland is of interest, since it has been shown that animals deprived of that gland cannot be made glycosuric either by pancreatic extirpation, or by phloridzin, or by Bernard's puncture of the fourth ventricle.

Diabetic Coma and Acidosis.—The coma of diabetes has been ascribed to lipæmia, to anæmia, to poisoning of the blood by acetone (*acetonæmia*) or by amido-oxybutyric acid, or by β-oxybutyric acid.

The occurrence of certain organic acids in abnormal amount in the blood and urine has been termed *acidosis*, and at a certain point of accumulation of acid the symptoms of *acid intoxication* are produced. The alkalinity of the blood may be little reduced, and the urine always contains an excess of ammonia. It has been practically determined that the chief acid causing this condition is β-oxybutyric acid, and not lactic acid; but whether oxybutyric acid acts by a specific poisonous action, or by combining with soda and potash bases, which are thus locked up so as to be unavailable for the transport of carbonic dioxide, is still open to discussion.

The existence of acidosis is revealed clinically by the presence of the acetone bodies in the urine, the tests for which have been given (*see* p. 969), since both acetone and diacetic acid are derived from oxybutyric acid.

Starvation in general, and deficiency of carbohydrates in particular, are causes of acidosis. Apparently this is due to the fact that in the absence of carbohydrates, the fats are drawn upon by the system, and from their metabolism oxybutyric acid arises. But in diabetes even if carbohydrates are supplied they cannot be utilised, and hence in this disease, acidosis and acid intoxication may occur both in untreated cases and in cases under treatment, if the carbohydrates are withdrawn from the food too suddenly or completely. In the acetonuria of starvation very small quantities of carbohydrate food will remove the acetone bodies from the urine.

Acidosis, or at least the presence of acetone bodies in the urine, *acetonuria*, occurs in some other conditions, namely, poisoning by opium, poisoning by phosphorus, acute yellow atrophy of the liver, eclampsia, the recurrent or cyclic vomiting of children, and the vomiting of pregnancy. In three of these instances, as well as in starvation, fatty degeneration of the liver is found. Another

condition associated with acidosis is post-anæsthetic poisoning, or "delayed chloroform poisoning," in which persistent vomiting, restlessness, and excitement, delirium, drowsiness, and coma are the symptoms; and in which the liver is frequently fatty in a high degree.

Diagnosis.—There is little likelihood of mistaking diabetes mellitus for any other definite illness; but the presence of the disease may be overlooked, and the patient may be treated for a vague weakness and "debility"; or the possibility of diabetes underlying one of its complications, such as carbuncles, pruritus, phthisis, or coma, may be forgotten. In all obscure conditions of weakness and emaciation, as well as in the case of carbuncles or frequent boils, spontaneous or senile gangrene, pruritus vulvæ, balanitis, impotence, double cataract or double sciatica, the urine should be tested for sugar. It must be borne in mind that coma may occur as a result of diabetes in persons not known to be diabetic; and that in diabetics, abdominal pain, severe enough to suggest that a laparotomy is needed, may be the first symptom of the onset of coma.

In exceptional cases where the urine cannot be tested, the following method for the detection of sugar in the blood may be employed: In a narrow test-tube are placed 40 cub. mm. of water, then 20 cub. mm. of the blood to be tested, then 1 cub. cent. of a watery solution of methylene blue (1 in 6000), and finally 40 cub. mm. of liquor potassæ (B.P.). Another tube is similarly prepared with healthy blood instead of the suspected blood, and the two test-tubes are placed in a beaker of water, which is then kept boiling for four minutes. At the end of this time the saccharine specimen has lost its blue colour and become dirty yellow, while the normal specimen remains blue or bluish-green (Williamson).

Prognosis.—The gravity of the prognosis has already been mentioned. Especially in young persons the disease tends to a rapid termination; while in the aged life may be more prolonged. When the disease has lasted some time, phthisis is very likely to develop; and in all cases the risk of rapid death from coma or pneumonia must never be lost sight of. The presence of diacetic acid in the urine shows the existence of acidosis, which may soon be followed by acid intoxication.

Treatment.—While the causes of a temporary glycosuria can be removed, the cause of a definite diabetes is either too obscure or too much dependent on irremediable conditions, such as pancreatic disease, to be dealt with. There remain as the chief indications for treatment the removal from the diet of all starchy and saccharine substances, and the use of certain drugs, such as opium, which appear to have a decided influence upon the formation of sugar.

The consideration of the diet is of the first importance. Nearly all cases are benefited, and some are for the time cured, by the avoidance of carbohydrates. For this purpose one should exclude entirely bread, whether toasted or not, potatoes, sugar, fruit, pastry, and farinaceous foods; and the diet should consist mainly of meat

with fat, eggs, green vegetables, and tea with cream, or a very little milk. Beer and sweet wines are to be avoided, but dry sherry, sauterne, or weak brandy and water may be taken if stimulants are desired. The deprivation of bread is most seriously felt by the patients, and various substitutes have been attempted, in which the nitrogenous elements are present and the starch is, as far as possible, excluded. Thus there are gluten bread, protene bread, casoid and casoid meal bread, bran biscuits, and almond biscuits; but, unfortunately, they are often very unpalatable, and are not always free from starch. It is an important question how far it is wise to put a patient suddenly on a diet which need not be less abundant than in health, but which is so greatly modified; and also whether carbohydrates should be absolutely excluded. The majority of cases bear very well the substitution of gluten and other special brands for ordinary bread, and are made worse if by any lapse they are allowed to take wheaten bread again. The treatment may be begun by excluding all starch and saccharine foods (potatoes, sugar, fruit), except a small quantity of bread; and then after a few days this may be exchanged for the gluten or almond substitutes. When the sugar has been reduced to its lowest point and maintained there for some time, carbohydrates may be cautiously resumed, and their effect on the sugar excretion watched.

Attention may be directed to a few special points. Fat is not harmful, and may even be beneficial; it can be taken as fat of meat or cream, or as cod-liver oil. Green vegetables are harmless, but blanched vegetables, such as celery, endive, and the white stalks of cabbages and lettuces, yield sugar. Animal soups may be taken, but must not be thickened with flour. Saccharin may be used instead of sugar to sweeten tea. The livers of animals contain glycogen, and should not be eaten; nor should oysters and other mollusca, and the interior of crabs and lobsters for the same reason. Lævulose may be taken in quantities up to 1½ ounces daily without increasing the glycosuria. Potatoes contain 15 to 20 per cent. starch, which is much less than bread; they are a useful food where a small quantity of carbohydrate is allowable. Toast contains as much carbohydrate as untoasted bread.

The following list of what may and what may not be eaten or drunk will show that there is a very considerable range allowed. The patient *may eat* butcher's meat of all kinds (except liver); ham, bacon, and tongue; poultry and game, fish of all kinds, fresh, salted, or cured; real turtle, mock turtle, and ox-tail soup; beef-tea; other broths and soups not thickened; essence of beef mutton or chicken; meat juice; pure meat lozenges; a very little onion, garlic, or shallot for flavouring broths, soups, or made dishes; bunch greens, spinach, watercress, mustard and cress; green lettuce picked from the white stalk; mushrooms, cucumber, vinegar; oil; pickles; eggs dressed in any way; cream cheese, cheese, butter and cream; gluten bread; almond biscuits; bran biscuits; cocoa-nut biscuits; blancmange, and custard made with cream (not milk)

and without sugar; jelly, flavoured but not sweetened; savoury jelly. Unless the very strictest dieting is required, celery and radishes may be allowed in moderation; and turnips, broccoli, cauliflower, Brussels sprouts, asparagus, seakale, vegetable marrow, and French beans may be taken if first boiled in a large quantity of water.

He *may drink* tea, coffee, cocoa from nibs; Liebig's extract of meat; cream; milk only sparingly, but more liberally sugar-free or "diabetic" milk; soda or potash water; Vichy, Vals, Royat, German seltzer, or Apollinaris water; dry sherry, claret, dry sauterne, burgundy, chablis, hock, brandy, or old whisky.

He should *not eat* sugar or starch in any form; ordinary brown, whole meal, or aerated bread, either plain or toasted; rice, arrow-root, sago, tapioca, ordinary macaroni or vermicelli, semolina, or any other farinaceous preparation; potatoes, carrots, parsnips, tomatoes, peas, broad beans, scarlet runners, cabbage, artichokes, endive, beetroot; pastry and puddings of any kind except those stated above; fruit of any kind, fresh or preserved; preserved ginger; oysters, or the interior of crabs, lobsters, &c.; any kind of liver; milk, except in small quantity.

He should *not drink* beer of any sort; sweet or sparkling wines or liqueurs; lemonade or any sweetened aerated drinks.

In the worst cases, or composite diabetes, even a rigid abstention from carbohydrates may fail to stop the excretion of sugar, which is then provided by the proteins either of the food, or of the body itself: and it may be found that under such a diet, when the sugar has reached a limit, below which it will not go, the patient is no longer benefited, and even loses more flesh and strength. In such cases it may be necessary to supply a certain amount of carbohydrates. A limitation of the proteids may then be also tried; or a diet of green vegetables, fat, cream, and eggs may be given for a period of a few days, and repeated at intervals.

Certain drugs have an undoubted influence in controlling the excretion of sugar in diabetes, but the diet must always be restricted at the same time. The drug that has the greatest influence is opium; it should be given in doses of ½ to 1 grain twice or three times a day, and gradually increased until, if necessary, it reaches 4, 5, or 6 grains in the twenty-four hours. Under such treatment, the sugar may entirely disappear and the patient regain flesh or strength; and if this condition is maintained, carbohydrates may be gradually allowed, and the opium may be slowly reduced to smaller and smaller doses.

The alkaloids of opium may be separately employed. Codeia is given in ½-grain doses three times daily, gradually increased to a daily administration of 20 or 30 grains. Morphia may be pushed gradually to a daily dose of 5 or 6 grains if required. Its effect upon the excretion of sugar is greater when taken by the stomach than when injected subcutaneously; but its influence on the sensorium is greater in the latter case (Mitchell Bruce). Small doses

of nux vomica will often neutralise the constipating effects of opium and its alkaloids.

The alkaline carbonates seem to be beneficial, and many patients are sent to drink the natural waters of Carlsbad, Vichy, or Neuenahr: at which health resorts they also, no doubt, benefit by bodily exercise, or by relief from business and mental worries. Good results have been observed from sodium salicylate, aspirin, and salol. Secretin, or an acid extract of duodenal mucous membrane, has been given by the mouth with the view of stimulating the pancreas; and it appears to have been at least temporarily successful in a few instances (Moore and Abram). Scrapings from the mucous membrane of the pig's small intestine are mixed and pounded in a mortar with an equal quantity of dilute hydrochloric acid (·4 per cent.), boiled for five minutes, and then nearly neutralised with solution of soda. The dose is from $\frac{1}{2}$ to 1 ounce three times daily. Other remedies have been vaunted from time to time—*e.g.*, carbolic acid, jambul seeds, uranium nitrate.

Of the complications, pruritus and eczema will probably improve as the sugar is diminished; borax ointment is a good application for the former. For the treatment of diabetic coma a vigorous alkaline treatment should be employed; thus, sodium bicarbonate ($\bar{3}$j to $\bar{3}$ij) may be given internally in milk, or the same quantity in solution by the rectum, or 2 or 3 ounces in 50 or 60 ounces of water injected into the connective tissues up to an ounce or more of the carbonate in twenty-four hours. And the internal or rectal treatment may be begun on the first indications of drowsiness. In default, or in addition, one may give diffusible stimulants, such as brandy, ammonia, ether, or camphor, and place hot bottles to the feet and legs, or put the patient in a hot bath, or inject hot normal saline solution into a vein. The preceding constipation tempts one to use purgatives, but they are often entirely inoperative.

FUNCTIONAL ALBUMINURIA

It has been already shown that albuminuria may occur in a number of morbid conditions, of which nephritis and renal degenerations, acute illnesses, infectious diseases, and venous congestion are the most important. Indeed, in the majority of cases in which albuminuria is found, the cause can be referred to one of these groups. But it is occasionally present in persons who appear to be in perfect health, or, at any rate, in working health. It may occur without any apparent cause, or it may be brought on by an exercise of function which in other individuals is quite harmless—*e.g.*, a full meal, especially of albuminous food, muscular effort, or exposure to cold. It may be of short duration, quickly passing away to recur from time to time, or it may persist for months.

Different cases of this disturbance have been described under the names physiological albuminuria, albuminuria of adolescents,

intermittent albuminuria, remittent albuminuria, cyclic albuminuria, postural albuminuria, and functional albuminuria.

The first of these is inapplicable. Numerous observations have been made on the presence of albumin in the urine in groups of individuals, such as, infants from one to six days old, schoolboys, bank clerks, soldiers, or workers in factories; and invariably albumin is found in a certain percentage, which varies from 5 to 30. In life-insurance practice it is common experience that a large proportion of applicants between eighteen and thirty years of age, believing themselves to be perfectly well, have a small quantity of albumin in the urine. These facts do not show that albuminuria is physiological: and it is probably due in these cases to conditions which, though not interfering with practical working health, are still deviations from the absolute normal. The term functional albuminuria has too wide an application, as it may fairly include albuminuria in heart or lung disease, where the kidney, at least in early stages, is as yet uninjured. Others of the above names indicate that the albuminuria is not persistent or that it varies with changes of posture or with exercise; and hence imply that it is not due to structural disease of the kidney: but the albuminuria of granular kidney is not necessarily always present, and certainly the quantity of albumin in many forms of nephritis may be influenced by physical conditions.

The term albuminuria of adolescents represents a fact of great importance, the explanation of which is still under discussion.

The albumin is as a rule small in quantity, sometimes a mere trace; but occasionally it is more abundant. Casts are often not to be found, even with the help of the centrifuge, unless the amount of albumin is considerable. The pulse is generally soft and the heart and its sounds are normal.

The following are different forms of albuminuria occurring mostly in young people, in good or moderately good health.

Athletic Albuminuria.—Albumin in small, decided, or even considerable amount is constantly found in the urine of those who have recently rowed or run a race, or have undergone other excessive physical exercise. There is no doubt that nearly all these cases recover, and that it is only a temporary disturbance, indicative of the vascular stress occasioned by physical effort.

Dietetic Albuminuria.—The chief cause is the ingestion of large quantities of albuminous food, such as eggs; in some persons any excess of food. Albumin is occasionally associated in the urine with crystals of calcium oxalate, which may possibly irritate the kidneys, and thus cause the passage of albumin; and such excess of oxalates may be produced by particular kinds of diet (*see* p. 892).

Orthostatic Albuminuria.—This includes the cases described as postural albuminuria, and probably also the cystic, remittent, and intermittent forms. It occurs in persons in good health, more often youths or young adults; and it is characterised by the presence of albumin at certain times of the day, while it is absent at others.

Thus, in the early morning it cannot be detected; it is present from about 9 A.M. to 5 or 6 P.M., and again disappears from the urine passed at night. The albuminuria is obviously determined chiefly by the assumption of the erect position, and by the accompanying exercise during the day; and it disappears as a result of the recumbent position at night. The amount of food taken has no influence upon it. Pavy observed that pure serum-albumin was not present in these cases, but a mixture of serum-albumin with alkali-albumin, precipitable by the organic acids, such as citric. In some of these cases it appears that the important factor is not the exact posture in itself, but the presence of lordosis, in consequence of which there is pressure by the spine upon the kidneys or the renal veins in the erect posture, which is relieved in recumbency. Such cases have been classed as *lordotic albuminuria*. In these persons the same effect has been produced by placing a cushion under the loins, when they are still recumbent.

Neurotic Albuminuria.—Disturbed innervation may be the cause of transitory albuminuria—*e.g.*, mental anxiety and prolonged study; and perhaps in this way may arise cases which are due to masturbation.

Paroxysmal Albuminuria.—This may undoubtedly occur as a phase of paroxysmal hæmoglobinuria, in which the hæmoglobin breaks up into hæmatin and globulin (*see* p. 855). The patients have malaise, a sallow tint, and subsequently albuminuria; the urine contains at the same time excess of urea and of urobilin.

Pathology.—The explanations of these varieties of albuminuria are still much discussed; and they are likely to vary with the views held as to the pathology of albuminuria in general. Abnormal diffusibility of albumin, increased pressure in the glomeruli (as from exercise or the erect posture), and vasomotor paralysis from modified innervation, are relied upon to explain some cases. Wright found a diminished coagulability of the blood in many cases of functional albuminuria; and giving calcium lactate to increase this property, he effected a diminution of the albumin. But these results want confirmation.

Some think that all functional cases are orthostatic, and that persistence of the albumin in spite of recumbency proves an origin in nephritis. But excluding the athletic cases, which appear to be practically normal, and the dietetic cases, in which probably assimilation is at fault, it is certain that a very large proportion of the individuals in which adolescent albuminuria occurs are not perfectly sound. They are often pale, thin, perhaps overworked, or they have recently grown rapidly, or bolt their food (Goodhart), or suffer from headaches. Some physicians connected with banks and similar institutions state that the subjects of adolescent albuminuria break down more readily, and are more frequently on sick leave, than their fellow-clerks. This suggests feebleness of tissue allowing transudation through membranes, or some other incapacity to resist the normal strain of working life.

Diagnosis.—In order to distinguish these cases from the graver instances of Bright's disease, a single examination of the urine is seldom sufficient. If albuminuria is discovered, as it often is, in the routine examination of the urine for life insurance, or admission to a school, a city office, or to the public services, where the individuals are presumably healthy; or if it is found in a young person who is at most only a little languid and out of sorts, a diagnosis of Bright's disease should not be hastily made, but the urine passed at different periods of the day should be tested to see if the albuminuria is determined by any of the factors—diet and position—alluded to; and especially to see if it is at any time absent. In chronic Bright's disease the quantity of albumin fluctuates during the day, and sometimes it is increased by exercise and diminished by rest; but the entire absence at night and during the early morning is very characteristic of the functional forms. At present, also, it is probably not safe to assume, because there are no casts and no high tension, and the patients ultimately get quite well, that they cannot have suffered from a mild nephritis.

Prognosis.—The future of these cases is on the whole favourable, and in the large majority the albuminuria entirely disappears, though it may persist for two or three, or even seven years. And undoubtedly very few develop into cases of chronic renal disease.

Treatment.—Where the cause can be recognised, such as diet or exercise, this should be altered or avoided. Where calcium oxalate seems to be responsible, dilute nitro-hydrochloric acid may be given, and meat, excess of vegetables, and wines should be excluded from the diet. In cases of orthostatic albuminuria, rest and milk diet may temporarily keep the albumin out of the urine, a result attributed by Wright to the calcium salts in the milk. But if the albumin is slight, and where the health is maintained, no treatment seems necessary other than the continuance of good hygienic conditions, and the preservation from undue strain of all kinds. The urine should be watched from time to time, and any other symptoms that arise must be met by suitable remedies.

BACILLURIA

The presence of bacilli in the urine has been more than once referred to. Thus in typhoid fever the urine may contain Eberth's bacillus (*see* p. 49), and in tuberculosis of the kidney the tubercle-bacillus is found. In other cases staphylococci, streptococci, the *Bacillus pyocyaneus*, and the *Bacillus proteus* have been seen. Of special interest is the invasion of the *Bacillus coli communis*, which frequently happens spontaneously, or from causes which are difficult to understand; it is often at any rate a primary lesion—that is, such inflammatory lesions of the tissues as are found to accompany it are its results and not its causes.

Ætiology.—It occurs at all ages, but it is especially frequent

in infants under two years of age. At all ages, females are much more frequently affected than males, and in adults pregnancy is a common antecedent. There are three possible means of access of the bacillus to the urine; one is from the colon through the tissues, a second is by means of the blood-vessels, and the third, which the frequency in females suggests as the most common, is that by the urethra. To this last method of introduction no doubt the use of the catheter sometimes contributes.

Condition of the Urine.—The urine is often clear when passed, and has a specific gravity of 1010 to 1015. On standing, it deposits a layer like mucus at the bottom, above this there is a haze or turbidity, and at the top a clear layer: or the urine is entirely turbid, and the turbidity does not clear on standing. The urine is always *acid*, and it does not become clear either with heat and acetic acid or with alkalies. Moreover, it has a peculiar offensive odour, which is not ammoniacal, but is probably due to sulphur compounds (methyl-mercaptan and sulphuretted hydrogen). A small amount of albumin may be present, and on examination with the microscope there are generally some pus-corpuscles, a few red blood-cells, and numerous bacilli. These can be shown by centrifuging the urine, making a film of the deposit, and staining with methylene blue. Their nature can of course be further demonstrated by cultivation and bacteriological tests. In advanced cases pus-cells and bacilli are more numerous.

Symptoms.—Bacilluria may be discovered in persons without any symptom at all: but at some time or other it is likely to cause either local or general disturbances. The local disturbances are due to irritation, and later inflammation of the urinary passages, the bladder, urethra, the pelvis of the kidney, and ultimately the substance of the kidney, constituting a suppurative nephritis. Thus, in small children incontinence of urine and frequency of micturition are frequent: and this may develop to a definite cystitis, in which fever may be absent, and the patient may not be ill. In a later stage, there is not only cystitis, but pyelitis as well, and the patient has pyrexia, with perhaps rigors. In a more advanced condition still, when the kidney is involved, there are all the usual signs of nephritis, namely, increased albuminuria, tube casts, severe pyrexial illness, prostration, uræmia, and a fatal termination.

It is probable that some hitherto unexplained cases of prolonged pyrexia in children are due to bacilluria of this type: and cases have occurred in which gastro-intestinal and cerebral symptoms of a severe type have been due to this kind of infection.

Diagnosis.—This is based on the physical and bacteriological examination of the urine, and can give rise to little difficulty so long as it is remembered that obscure conditions of fever, or even cerebral symptoms, as well as local evidences of cystitis and pyelocystitis, may be due to this infection.

Treatment.—In early stages the disinfection of the urine may be attempted by the administration of urotropin (10 grains three

times a day) or helmitol; or by washing out the bladder with a solution of silver nitrate (one to a thousand or two thousand). In more advanced cases the treatment by vaccines of bacillus coli has given good results; if streptococci or staphylococci are present a mixed vaccine should be employed. Thompson believes that it is sufficient to render the urine fully alkaline by adequate doses of potassium acetate or citrate.

PNEUMATURIA

A brief notice may be given of this rare condition, the presence of gas in the urine. It may obviously occur in the event of a communication between the rectum and the bladder; but apart from this, gas may be formed as the result of fermentation. In diabetic urine the glucose may be fermented by yeast-cells, or by the bacillus coli communis, producing carbon dioxide. Albuminous urine in cystitis, and apart from diabetes, may be fermented either by the bacillus lactis aerogenes or the bacillus coli communis. The gases yielded are carbonic monoxide and hydrogen. To remove this condition the glycosuria in the one case and the cystitis in the other should be treated.

CHRONIC INTOXICATIONS AND THE EFFECTS OF HEAT

ALCOHOLISM

Under this term are included the results of the more or less continued use of alcohol, not the immediate effects of a considerable overdose, known as drunkenness or intoxication. The symptoms of the latter are familiar, and usually subside as the alcohol passes through the system. Occasionally, however, from the rapid imbibition of large quantities of spirit in those unaccustomed to it, a fatal result may ensue. It is preceded by unconsciousness, with pupils sluggish or fixed, dilated or contracted; small pulse, cold clammy skin, stertorous respiration; and sometimes delirium or convulsions.

DELIRIUM TREMENS

This commonly occurs in those who habitually drink freely, who may have been not infrequently drunk, and who have recently been taking unusual quantities continuously for some days. It is often indeed stated that the patient had left off drinking for two or three days before the symptoms came on; but as a distaste for drink is sometimes one of the first manifestations of the disease, it is probable that this is an explanation of the apparent anomaly. Delirium tremens is also sometimes determined in habitual drinkers by some severe shock, such as the fracture of a bone, or the onset of pneumonia, erysipelas, or other acute disease, without evidence of any amount of drinking beyond the daily average.

Symptoms.—The first symptoms are disturbed sleep, restlessness, irritability, and loss of appetite; and with the disturbed sleep there are unpleasant dreams and some wandering. In the morning the patient may be more rational, but the delirium returns at night; the next day the delirium continues, and shows its characteristic features. The patient is constantly talking, addressing either those about him, or imaginary persons whom he supposes to be present. He talks on his business, or on other affairs, frequently changing from one subject to another. He may be recalled to his senses for a moment, but soon relapses. In the pursuance of his thoughts he may try to get out of bed, or pull the bedclothes about. With this there is very marked tremor; the hands shake with quick oscillation whenever he moves them; the lips and tongue tremble when he speaks. As the condition gets worse he has very definite hallucinations and illusions. Objects in the room or figures on the

wall-paper are conceived to be animals or insects: he is constantly seeing cats, dogs, rats, mice, or blackbeetles running after him or crawling about the bed; he looks under the bed, or behind the curtains, or peers behind any bystander, and is suspicious of injury from those about him. He may, under some such false impression, strike those who are near; but, as a rule, he can, with a little firmness, be controlled, and it is often easy to turn his thoughts for a moment in another direction.

Other symptoms accompany this mental and muscular disturbance. The face is flushed, the conjunctivæ are suffused, the tongue is thickly furred and becomes dry, as the disease progresses. The pulse is quick, soft, at first full, afterwards small and feeble: the temperature rises to 102° or 103°, and there is occasionally hyperpyrexia (106° or more). The skin is generally moist or even profusely perspiring; and the urine is accordingly dark-coloured, scanty, and of high specific gravity. It may contain a small quantity of albumin. According to Gowers there may be indications of early optic neuritis.

About the end of the third day, with considerable constancy in favourable cases, some improvement begins to show itself. Hitherto sleep has been entirely absent, but now the patient falls into a slumber which may last eight or ten hours, and he wakes much refreshed, the delirium and trembling are less, and gradually improvement takes place in all respects. In more serious cases the face loses its colour and becomes pale and earthy, the pulse is quick and feeble, the delirium is less active, and the patient more prostrate; he lies on his back muttering, and semi-comatose. Finally death takes place, preceded, it may be, by convulsions or hyperpyrexia. In some instances the symptoms are comparatively mild, and subside in a day or two; in others, the long-looked-for sleep is not directly curative, but is repeated more than once before recovery is ensured.

Death occurs from exhaustion; from cardiac failure, and this is sometimes sudden; or from pneumonia.

Morbid Anatomy.—The changes are not very pronounced; some congestion of the cortex of the brain, of the bases of the lungs and of the kidneys, may be all that can be found. Thickening and opacity of the membranes of the brain is a chronic condition not responsible for the acute symptoms.

Diagnosis.—It may be simulated by *meningitis*, and apparently by *general paralysis of the insane*. The former is generally distinguished by early convulsions, or later on by paralysis, sometimes, but not always, by headache, or by the duration of the symptoms. In the latter, a carefully studied history ought to help to a right conclusion. In all cases of delirium tremens it should be remembered that pneumonia or fever may be present.

Treatment.—The patient should be kept as quiet as possible in a darkened room undisturbed by visitors. If he is restless and gets out of bed, male attendants are desirable who may with a little

management keep him under control. Extreme violence may require a strait-waistcoat, or a sheet stretched over the trunk, legs, and arms, and fixed to the sides of the bed; or broad padded leather bands, by which the shoulders, wrists, and ankles can be fixed down to the bedstead, thus leaving the chest free from restraint. These methods should, however, if possible, be avoided. The likelihood of the patient doing injury by getting at knives or forks, by drinking lotions or liniments, or by jumping out of the window must be borne in mind. Food must be given every two or three hours in small quantities; it may be milk and beef-tea, and it is better if it can be peptonised by the use of liquor pancreaticus or similar preparations. The use of drugs has to be approached with great caution. Narcotics seem to be called for, and potassium bromide, sodium bromide, chloral, opium, morphia, and sulphonal are those which may be employed. But there is, in delirium tremens, a remarkable resistance to the action of such drugs, and the fear is that, finding the usual doses are inoperative, one may proceed to larger and larger quantities, which remain in the body, and are only too dangerously effective as the disease is subsiding. Fifteen or twenty grains of chloral may be given every six hours; or a quarter of a grain of morphia or $\frac{1}{150}$ grain of hydriodate of hyoscine may be injected, and repeated at the same interval, if required. Stimulants are better avoided; but they may become absolutely necessary in the stage of prostration, when ammonia, ether, or alcohol may be given.

CHRONIC ALCOHOLISM

The effects of chronic alcoholism are seen most markedly in various nervous symptoms, in impaired digestion, and in cirrhosis of the liver; it no doubt also contributes to fatty and to atheromatous degeneration of the arteries, and to diseases of the kidneys. These different parts of the body may be affected separately or together; but, as a fact, the term chronic alcoholism is more commonly applied to those cases in which the liver alone, or the kidneys alone, are the seat of disease. In all cases it is important to observe that it is not drunkenness or intoxication which leads to this condition, but the constant imbibition of considerable quantities of beer, wine, or spirits, possibly without the individual getting drunk on any single occasion. The daily amount of alcohol and the duration of the drinking habits required to produce this effect in different persons are very variable.

Symptoms. *Nervous System.*—Muscular tremor is one of the first indications. The hands are unsteady, and the tongue trembles when it is protruded. The patient is restless and irritable, he sleeps badly, and wakes unrefreshed, with a feeling of prostration that tempts him to take stimulants at once. Sinking feelings are frequently complained of, which require, according to the patient, to be met by fresh doses of the favourite drink. As things

get worse, the patient is unequal to any sustained mental effort; even the simplest business transaction must be preceded by a glass. Sensory disturbances may be also present, such as buzzing or rushing in the head, vertigo, muscæ volitantes, flashes of light, or diffused headache. There may be severe neuralgic pains in the legs, possibly the early signs of neuritis. In later stages the mind is seriously involved. Judgment, intellectual capacity, volition, and the moral sense are all weakened. The patient becomes hesitating and vacillating, unable to follow out any definite line of action, but unscrupulous in his attempts to get stimulants at all times. Multiple neuritis (*see* p. 217), epileptic fits, irregular paralysis or anæsthesia, and some forms of insanity, which may be mania, melancholia, or dementia, also occasionally result from continued excess.

Gastric Disturbance.—This is generally shown quite early, coincidently with the first nervous symptoms. The patient vomits in the morning directly he rises from bed, he is quite unable to eat any breakfast, and his appetite generally is deficient. The tongue is covered with thick yellowish fur, and the breath is fœtid. The eyes are suffused, and the face may be tinged with yellow.

In course of time the minute venules of the cheeks become dilated, the nose is red and thickened, and sometimes rosacea develops. The face becomes more and more bloated, and the blotching with dilated venules is more marked. In this stage, or a little earlier, the liver may be found to project 2 or 3 inches below the costal margin, and the urine frequently contains albumin. Dilatation of the heart with its consequences (*see* p. 617) occurs in a small proportion of cases.

Finally, in many cases the obesity, which is the early result of alcoholism, gives way to the converse condition; and it is not uncommon to see the patient with wasted chest, arms, and legs, and the abdomen large from retained fat, or cirrhosed liver and ascites; the feet are perhaps œdematous, while the urine is scanty (*see* Cirrhosis of the Liver, Chronic Interstitial Nephritis, and Gout).

In addition to the diseases directly resulting from alcohol, these patients are liable to succumb with great rapidity to any acute illness, such as pneumonia, erysipelas, or typhus.

Pathology.—In chronic alcoholism the membranes of the brain are commonly opaque and thickened, and the convolutions are shrunken and atrophied. The changes in neuritis, hepatic cirrhosis, &c., are described elsewhere.

Treatment.—The one essential is the abstention from alcohol in any form, and it is desirable that this should be immediately enforced, with no attempt at diminishing the quantity day by day. The patient will advance all sorts of excuses as to why he should not give it up at once, but they should not be regarded. No drug is of any value as long as the drinking is continued, but the craving may be perhaps diminished, and the patient generally benefited, by tonics, such as quinine, cinchona, nux vomica, and cod-liver oil.

To obtain sleep, bromide of potassium, chloralamide, trional paraldehyde, hyoscyamus, Indian hemp, and rarely morphia may be employed. Where patients will submit to the restraint, the *régime* of a hydropathic establishment is eminently suitable.

LEAD-POISONING

(*Plumbism*)

This occurs among those who have to do with lead or lead salts, either in the preparation of those substances, or in industries in which they have to be used. The latter are especially printing, plumbing, type-founding, type-setting, glazing of china and pottery, and glass-polishing. It also arises accidentally as the result of impregnation of drinking water with lead from the cisterns in which it is stored, from taking snuff which has been packed in lead paper, from the use of hair-dyes, or otherwise. Lead may thus enter by the alimentary canal—the most frequent way—by the skin, or occasionally by the respiratory mucous membrane. Like alcohol and other poisons, it acts very differently on different individuals: some men are attacked with characteristic symptoms within a few months of entering a white-lead factory; others may work in it for years with impunity. The tendency to be attacked is increased by starvation, ill-health, exposure to cold, and indulgence in drink (T. Oliver).

Symptoms.—The effects which it produces on its victims are—(1) colic; (2) paralysis and other nervous symptoms; (3) "blue line" on the gums; (4) anæmia; (5) changes in the urine; (6) interference with the function of reproduction.

(1) *Lead Colic.*—This is a form of intestinal colic. After a few weeks of impaired health, as shown by headache, sick feeling, and pallor, the patient is seized with severe spasmodic pain at or around the umbilicus; the abdominal muscles are contracted, and the pain is rather relieved by firm pressure in many cases, but often the abdomen is tender, and it is generally retracted. Sometimes there is vomiting, and the bowels are nearly always confined. The pain diminishes for a time and then recurs; and is generally relieved in the course of one to three or four days. It is probably due to spasmodic contraction of the intestine, with some diminution of secretion from the intestinal mucous membrane; but exactly how these are caused still remains obscure. Oliver notes that during colic the pulse is slow and hard, and the urine scanty. *Vomiting* is sometimes a troublesome symptom before the occurrence of colic.

(2) *Lead paralysis* has been already described under multiple neuritis (*see* p. 219).

Cerebral Symptoms. Saturnine Encephalopathy.—These may be in the form of hemiplegia, or of hemianæsthesia. More severe cases, not infrequently fatal, occur in which convulsions, delirium, and

coma, with, perhaps, optic neuritis and some fever, are the symptoms. Such cases often run a very acute course, and appear to be more frequent in females (Oliver). Anæmia is the first symptom, and then colic, headache, vomiting, diplopia, or defective vision from optic neuritis. In a few days the patient is convulsed, becomes comatose and dies. In other instances there is mental disturbance, amounting to insanity, which is either acute mania or melancholia, or progressive mental failure and muscular weakness, with, perhaps, convulsions.

Ocular lesions and symptoms are frequent, and include optic neuritis with or without hæmorrhages, neuro-retinitis, primary and consecutive atrophy, inequality of pupils, diplopia, and a bilateral amblyopia without change in the fundus, similar to uræmic amaurosis.

(3) The *blue line* on the gums, or *lead line*, has mainly a diagnostic importance, showing that lead has been taken into the system. It is a dark slate-coloured or black finely-dotted line, which forms in the gum close to the teeth, and consists of a deposit of sulphide of lead in the tissues around the vessels; this results from the union of lead with sulphur provided by albuminous substance (partly contained in "tartar") at the edge of the gum. Where teeth are absent there is no blue line, and if the teeth are kept exceptionally clean, or the gum lies close up to the teeth, the blue line is absent. In such cases it may be seen only in portions of gum rising between the teeth. Sometimes a blue patch is seen on the inside of the cheek opposite a decayed tooth. The blue line may exist without any other symptoms of plumbism; it persists for from eight days to three months, or much longer, according to some others, after all entry of lead into the system has ceased.

(4) Sufferers from lead-poisoning are generally *anæmic*, often remarkably so, with a sallow earthy look. The red corpuscles are diminished in number, and contain less than their normal amount of hæmoglobin (chlorotic type). The anæmia is often the earliest indication of impregnation with lead.

(5) Albuminuria is a result of lead-poisoning in two or three ways. It may occur temporarily in lead colic: and it is often the result of more definite renal change, in the form either of tubal nephritis, as in cases of encephalopathy (Oliver), or of granular kidney in more chronic conditions of plumbism. The relations of lead, gout, and granular kidney are very close. Gout, at least in the south of England, is common in those who suffer from chronic lead-poisoning, which causes, it is said, diminished excretion of uric acid. And it has also been observed that sufferers from gout are very readily affected by lead.

The urine also often contains indican, and lead itself in combination.

(6) Lead-poisoning affects the functions both of menstruation and of gestation. Menorrhagia is very common, but occasionally there is amenorrhœa. In pregnant women there is a large pro-

portion of miscarriages and still-births; and of children born alive many are under weight, and many die within a year. Amongst those who survive, convulsions, imbecility, and idiocy are above the average proportion.

Pathology.—Lead has been found in various organs, namely, the brain, liver, and spleen, also in the bones; but in very small quantity. It obviously interferes in some way with the formation of blood, as shown by the anæmia, and profoundly modifies other metabolic processes in the body, as seen by its relation to uric acid. The cæcum and colon have been found blackened by sulphide of lead. The paralytic symptoms are mainly due to a neuritis, which has been found most marked in the intra-muscular twigs; it is less so in the larger trunks, and is usually absent in the parts near the nerve-roots. In a few cases atrophy of the nerve-roots has been seen, and in some changes in the anterior cornual cells, but these are probably secondary to the neuritis. In the muscles the usual degenerative changes are found (*see* p. 214).

Diagnosis.—This depends for the most part upon the history of lead-poisoning, upon the presence of a lead line upon the gums, and upon the discovery of lead in the urine. The history may have to be investigated with the greatest care, as lead may get into the system by the most unexpected means. If paralytic symptoms are present, a history of colic is often obtainable, and in nearly all cases the lead line is present, though in very cleanly persons it may only be found beneath the teeth, or possibly not at all. Double paralysis of the extensors of the forearms is probably due to lead; but if the small hand muscles are involved, the resemblance to *progressive muscular atrophy* is very close.

To detect lead in the urine, a small bag containing calcium sulphide may be suspended in it. The calcium sulphide will be blackened owing to the formation of lead sulphide.

Prognosis.—Typical wrist-drop often recovers, but it may be very slowly. Severe cerebral symptoms endanger life, but if death does not occur, recovery may be complete.

Treatment.—The patient should give up his occupation, or prevent in whatever way may be necessary the introduction of more lead into the system.

Colic generally yields readily to a full dose of opium combined with a simple purgative; an ounce of castor-oil with 15 or 20 minims of tincture of opium is a common and successful prescription. Sometimes it may have to be repeated; a strong saline purge (magnesium sulphate) may be given, or a drop of croton oil may be added to the castor-oil. Hypodermic injection of morphia may be used instead of opium. Hot fomentations or poultices should in the meanwhile be applied to the abdomen. Iodide of potassium is believed to hasten the elimination of lead, and may be given in 5-grain doses, three times daily, for two or three weeks afterwards, but it seems in some cases to have put into circulation with ill effect leadt hat was previously latent and harmless.

In *paralytic affections* the iodide should also be given, and the muscles may be galvanised with the continuous current; if after some time they react well to the faradic current, this should then be substituted. Massage may also be tried. For *acute cerebral attacks* Oliver recommends the inhalation of nitrite of amyl and lumbar puncture with removal of from one to three ounces of fluid.

Anæmia must be met by suitable ferruginous treatment in addition to what is necessary to prevent further impregnation, such as removal from work and the use of potassium iodide.

Prevention.—It is unnecessary to say much on this point. The protection of lead-workers is a matter for the employers of labour, or, failing them, for the State. It is obvious that cisterns or pipes used for drinking-water should not be constructed of lead, nor should recourse be had to this metal for the preservation of anything, such as food, drink, snuff or tobacco, which is to be afterwards taken into the system.

CHRONIC MERCURIAL POISONING

This is chiefly brought about by the inhalation of the vapour of mercury in those who work in quicksilver mines, in making barometers and thermometers, and in other manufactures. Many such persons are affected with the anæmia, salivation, and stomatitis which are the familiar results of overdosing with mercury used as a drug.

The characteristic of mercurial poisoning is the tremulous movements of the limbs and body known as "mercurial tremors," or "trembles." They come on suddenly or gradually, generally after exposure for some years to the metallic vapours. They affect the arms first, and then spread to the legs and the muscles of the rest of the body. The movements are at first brought out only by excitement, later whenever the patient makes any muscular effort, and finally they become constant, and even persist to a certain extent during sleep. The tongue is tremulous, and speech is hurried, abrupt and jerky. The patient may be quite unable to walk without support. In the earlier stages there is some resemblance to disseminated sclerosis, but there is no nystagmus; and if the movements become constant, paralysis agitans is, to a certain extent, simulated. Various cerebral symptoms, such as delirium, hallucinations, and mania, occur in extreme cases.

Recovery may take place if the disease is not too advanced.

Treatment.—Removal from the fumes of mercury is essential. Tonics and iodide of potassium are of most value as drugs.

CHRONIC ARSENICAL POISONING

This is less common than lead-poisoning, but it may arise in the following circumstances: Among persons employed in arsenic factories, and in industries involving the use of arsenical pigments; among persons using such coloured articles, as, for instance, certain gray and green wall-papers; occasionally from the use of large doses of arsenic in medical treatment; and in accidental impregnation of beverages with arsenic, such as occurred in the Manchester epidemic of peripheral neuritis, when an impure sulphuric acid was used in the process of malting.

Symptoms.—In the case of exposure to arsenical dust, the effects are manifest on the skin and accessible mucous membranes; when the arsenic is taken internally, the alimentary mucous membrane, the peripheral nerves, and the skin through the circulation are affected. In the first case the prominent features are irritation of the skin resulting in a form of eczema or dermatitis, which is seen especially in the warmer or moister parts, such as the axillæ, between the scrotum and thighs, at the edges of the nostrils and the eyes. Redness of the conjunctiva and smarting of the eyes, sore throat and irritation, with frequent hawking, are also present. Gastro-intestinal irritation follows, with perhaps sickness, diarrhœa, and abdominal pains. With this are often combined emaciation, weakness, muscular cramps, and frontal headache. The symptoms following ingestion of the poison in frequent small doses are often different from the above, and are seen especially in the production of peripheral neuritis (*see* p. 217), and in a disorder of the skin, of which pigmentation and thickening of the epidermis, especially on the palms and soles, or *keratosis*, are the prominent features. Transverse ridges and furrows appear on the finger-nails; and in acute cases curved white lines (*leuconychia*) have appeared showing interrupted nutrition at the time of the poisoning.

Treatment.—This consists in avoiding the cause, when the symptoms will subside. Peripheral neuritis may, however, persist for several months.

INSOLATION

(*Sun-stroke, Heat Apoplexy, Thermic Fever*)

Insolation occurs only occasionally in the hottest summers in the British Isles, but frequently in India, and other tropical parts. It has been generally regarded as the result of heat upon the body, though not necessarily caused by the direct action of the sun's rays. Thus the symptoms develop in barracks, workshops, and similar buildings, almost as often as in the open air. A moist atmosphere is more favourable than dry air, and the individual is predisposed to it by habits of intemperance in general, and by excessive exertion at the time.

Symptoms.—The symptoms are not always the same, and it has been usual to describe three forms: a syncopal, an asphyxial, and a hyperpyrexial form:

Syncopal or *Cardiac Form.*—The patient suffers from faintness, or complete syncope, nausea, or vomiting; the surface of the body is pale, cool, and moist, and the temperature is subnormal; the pulse is quick and feeble; the pupils are dilated. Death may take place from continued heart-failure, but recovery is frequent. In case of survival, fever, headache, pallor of skin, quick pulse and respiration may continue for some time, and the temperature may rise to a high level.

Asphyxial Form.—In this the symptoms come on quite suddenly—a true stroke. The same collapse and cardiac failure are present as before, but respiration fails as well.

Hyperpyrexial Form or *Siriasis.*—This often develops gradually, the early indications being weakness, restlessness, and sleeplessness, nausea, or vomiting, thirst and anorexia, giddiness or headache, hurried breathing or præcordial anxiety, and often profuse and frequent micturition. There may be incoherent talking, or a temporary state of delusion; the patient becomes unconscious, with laboured stertorous breathing, rapid feeble pulse, contracted pupils, and livid or congested face; and the temperature is found to reach 109°, 110°, or 111°. This may be succeeded by convulsions, suppression of urine, and death.

Anatomical Changes.—Very little is noted in the cases of rapid death from cardiac or respiratory failure. In the hyperpyrexial variety the blood remains fluid, the left ventricle is contracted from coagulation of its myosin, and the right ventricle is dilated; the lungs are intensely engorged, with hæmorrhage under the pleura, the cerebral meninges are congested, and it is said that there may be evidence of early meningitis. Acute parenchymatous degeneration of the neurons is described by Van Gieson.

Pathology.—This is still but little understood. It is apparently a result of the operation of external heat upon the central nervous system including the heat-regulating mechanism; and Van Gieson believes the intermediate link is the production of an autogenetic poison in the blood. According to Sambon, the syncopal form is merely a cardiac failure, which may occur in any country should the heat become extreme, whereas the hyperpyrexial form, or *Siriasis*, is really an infectious or even epidemic disease, limited to the tropical or subtropical countries in which it is endemic, but not determined solely by the effects of the sun's rays or a hot atmosphere upon the bodily tissues.

Prognosis.—The mortality is stated to be from 45 to 50 per cent.. and in cases that recover sequelæ are often observed, which may be, according to Fayrer, weakness from obscure structural change in the cerebrum, or chronic meningitis, epilepsy, defective memory, nervous irritability, headache, insanity, partial paraplegia, partial or complete blindness, or extreme intolerance of heat. The syncopal form is least likely to be attended with sequelæ.

Treatment.—Removal from the source of heat and the use of the cold douche are necessary in all varieties. In the syncopal form the douche must not be too long continued, and the condition of the pulse may require direct stimulation. In the hyperpyrexial form the temperature must be reduced as quickly as possible; if ice can be obtained, it should be put in the water used, or it may be rubbed directly over the body of the sufferer, until the temperature in the rectum has fallen to 104°; or iced-water enemas may be used. After this it will fall to the normal unassisted. Manson objects to chemical antipyretics, but recommends the subcutaneous injection of 40 minims of tincture of digitalis. For sequelæ, removal to a cool climate, and the use of iodide of potassium and counter-irritants are advised.

DISORDERS OF NUTRITION MOSTLY INVOLVING BONES AND JOINTS

The local diseases of joints and bones fall properly within the province of the surgeon; but general disorders involving many bones or joints may be considered in a work on medicine.

Multiple affections of joints are often inflammatory in nature, and result from various forms of infection—*infective arthritis.*

Multiple diseases of bones are, much more than those of the joints, referable to malassimilation or perverted nutrition; and are very obscure in their pathology, that is, though their ætiology may be plain, and their antecedents well known, there are many steps in the process of their causation still unrevealed.

INFECTIVE ARTHRITIS

It will have been observed that in several of the infectious diseases arthritis has been mentioned as a complication; the most common case of a multiple arthritis, namely, rheumatic fever, is now recognised as an infective disease; and it is believed by some that osteo-arthritis is equally due to microbic invasion. In the following diseases a multiple arthritis has been seen as complication or sequela: Typhus, typhoid, influenza, pneumonia (pneumococcal arthritis), septicæmia, pyæmia, gonorrhœa, dysentery, scarlatina, dengue, syphilis, both congenital and acquired, tubercle, and erysipelas. Whether these are due in every instance to the specific organism of the disease, or to a secondary infection, is not always clear. Smallpox is said to be followed sometimes by pyæmia, and sometimes by "rheumatism"; the former is no doubt secondary, but the inflammation called rheumatic may be specific.

Pulmonary osteo-arthropathy is another form of multiple arthritis, toxic in origin, and most often associated with suppuration. The multiple arthritis of certain nervous diseases, locomotor ataxy and syringomyelia, and that of gout can scarcely be placed in this group at present.

Infective arthritis may be a simple synovitis, which soon recovers; or it may result in fibrous ankylosis or in suppuration. In some diseases, such as rheumatic fever and scarlatinal synovitis, recovery is the rule; in others, like pyæmia, septicæmia, and pneumococcal arthritis, suppuration is frequent or invariable; in others again, like typhoid fever, influenza, and erysipelas, every one of the three results is sometimes seen. Surgical treatment is required in many

cases. The milder forms subside with rest; severer forms require splints and fixation; and where suppuration is recognised the joint should be opened and drained.

GOUT

In its typical form this is an acute arthritis, associated with the deposit of crystalline sodium biurate in the joint affected, and with the circulation of an excess of urates in the blood. In the majority of cases the feet, and especially the great-toe joints, are first attacked, whence the classical name *podagra;* but the joints of the hand (*chiragra*) and other articulations are subsequently, and much less commonly even first, affected. Certain symptoms in various parts of the body, and some morbid conditions of the organs, occur in the intervals between the attacks of articular inflammation. For these the terms *atypical, irregular, abarticular, metastatic, retrocedent,* and *visceral* gout have at different times been used. Of the visceral affections the most important is that form of chronic nephritis which is known as gouty or granular kidney (*see* p. 924).

Ætiology.—Gout is well known to be strongly hereditary, so that the descendants of a gouty stock are liable to outbreaks of the disorder at an early age and with comparatively little exciting cause. It is more common in men than in women, and is a disease of middle life or advanced age, though it does occasionally, in the hereditary cases just referred to, appear as early as the age of twenty; and it has been seen in boys who were only eight, nine, or twelve years old. It has been often regarded as a disease of the rich, from which the poor escape; but this is not entirely true, as the disease is often seen in its most typical form among hospital patients and others in poor circumstances. The influence of wealth is related to diet, which is the most important ætiological factor; and the ingestion of large quantities of food, especially of a highly nitrogenous kind, with abundance of alcoholic liquors, directly contributes to that condition of the blood and tissues which is the essence of gout. Of alcoholic beverages, malt liquors and the stronger wines like port and sherry seem to be more prejudicial than distilled spirits. The effects of dietetic excess are aggravated by a sedentary life; and as a rule, an occupation is prejudicial in proportion as it tempts to one or necessitates the other. Prejudicial also are occupations, which, like house-painting, type-founding, &c., expose the operatives to lead-intoxication, and hence increase in them the liability to gout (*see* pp. 924, 990). In those who are predisposed to it, or who have already had manifestations, an attack may be brought on by an aggravation of the dietetic excesses, or by any departure from the strictest regularity hitherto found necessary; by anxiety and mental worry; and sometimes by injuries.

Symptoms. *The Gouty Attack.*—In the majority of persons gout first shows itself by an attack of acute inflammation in the

metatarso-phalangeal joint of one great toe. Various premonitory symptoms are noted in different cases; in some it may be an unusual feeling of health or exhilaration; but more often they are such as the following: Mental depression; disturbed sleep; odd sensations, itching, or cramps in the limbs; tinnitus aurium; salivation, gastralgia, vomiting, or flatulence; alterations in the quantity and colour of the urine, which is mostly scanty and loaded with urates. These symptoms may have been troublesome for a day or two, when the patient is awakened, commonly about two o'clock in the morning, with pain in one great toe. The pain becomes worse and worse, and the patient finds it impossible to get ease. At the same time there may be a little chill, or even a rigor, and some fever. After some hours of excruciating pain, this at length abates, and the patient may fall off to sleep; when he awakes again he finds the affected joint red and swollen. It is exquisitely tender, the skin is tense and shining, and if it can be touched, pits slightly on pressure. The veins around it are slightly distended. During the day the patient may be free from severe pain, but towards evening there is a recurrence of all the early symptoms—that is, of severe pain and some febrile reaction—which remit towards morning, to return again the following night. The joint continues swollen, and the swelling extends in the cutaneous tissues some distance up the foot; the colour is a dull, dusky red. When at length the inflammation subsides, which it does in from five to ten or fourteen days, the skin desquamates in large thick flakes, and gradually assumes its normal colour. In exceptional cases one or two toe-nails may be shed; on the other hand, in mild attacks desquamation does not occur.

The general condition of the patient is one of slight febrile reaction, with more or less gastric disturbance. The temperature is not much raised; the thermometer may reach 101°, but is rarely so high as 102°, and then only for a short time. The tongue is thickly furred, and the patient has no appetite, but much thirst, nausea, and sense of distension at the epigastrium; the bowels are confined, and the motions are deficient in bile. Sometimes the sense of distension and the tenderness extend to the hepatic region.

In many cases during the first few days the urine is scanty, highly coloured, and of great density, but the uric acid is diminished by half; as the attack passes off, the uric acid increases, may exceed its average amount, and then subsides to the normal, while the urine has again acquired its usual quantity, colour, and density. The blood also, during the attack of gout, may contain an excess of uric acid combined as urates, which may be demonstrated by the thread experiment of Garrod. This consists in placing two drachms of serum obtained from a blister in a shallow watch-glass, adding ten or twelve drops of acetic acid, placing in the serum three or four fine threads, and setting it aside for thirty or forty hours at the ordinary temperature. At the end of this time, if the threads be examined under the microscope, crystals of uric acid will be found to have formed upon them. But these variations

of uric acid in the urine and the blood are not constant. In an acute attack there is leucocytosis with an unusual proportion of large uninuclear vacuolated cells without neutrophile granules, which may be myelocytes (Watson), and some excess of eosinophiles.

When an attack of gout is at an end, the patient often feels better than he has done for a long time before; and he is, as a rule, free from any reminder of his condition for a period of several months, or even two, three, or more years. His second attack may be in the same joint as the first, an almost exact reproduction of it; or it may occur in the opposite foot, or in one ankle, or in the wrist or hand. A third attack often comes at a somewhat shorter interval than that between the first and second; and the periods of repose diminish in length as time goes on. Ultimately a great many joints have been at one or other time affected, and with repeated attacks they undergo changes which result in considerable deformity, so that the old gouty subject becomes crippled in somewhat the same way as the sufferer from osteo-arthritis (*see* p. 1004).

When the disease has reached this stage it is in reality a *chronic gout.* If the small joints of the toes and fingers are often first affected, ultimately all the joints of the extremities may become the seat of gouty deposit, the shoulder and the hip less frequently. In the hand the joints are enlarged, are more or less fixed in different positions of flexion or extension, and in severe cases there is deviation of the fingers to the ulnar side of the hand. Similarly the foot may be fixed in a condition of talipes, or the knee or elbow in a flexed position. The swelling about the joints is often assisted by the existence of white deposits called *tophi,* which at first lie close under a thin shining skin, with dilated venules. But subsequently the skin may yield, and the creamy or chalk-like deposit may escape in small quantities at a time; or, more rarely, suppuration takes place around the deposit, and leads to its more rapid elimination. These tophi are not confined to the affected joints; they are seen under the skin of the fingers adjacent, in the bursæ (for instance, over the olecranon), in the tendons, and with considerable frequency in the cartilage of the helix of the ear. If the creamy juice from one of these deposits be examined under the microscope, it will be found to consist of innumerable minute acicular crystals, which are composed mostly of sodium biurate, with a small proportion of calcium urate or phosphate, and sodium chloride.

Irregular Gout.—The disturbances included under this head are very various, and consist of inflammatory lesions and functional disorders in many organs in the body. The former include gastric and intestinal catarrh, bronchitis, conjunctivitis, iritis, gouty urethritis (which is, according to Ebstein, a prostatorrhœa), phlebitis, and neuritis. According to some authors, cirrhosis of the liver may result from gout, independent of the alcohol which is so frequently indulged in by gouty individuals. Chronic nephritis (granular kidney) has already been mentioned: it is a frequent sequel of chronic gout, but the kidneys have been found in a condition of

granular nephritis with uratic deposits, while the joints were entirely free; and probably in other cases the kidneys are affected before the joints (primary renal gout, Ebstein). Some diseases of the skin, especially chronic eczema, are seen in connection with gout. On the side of the circulation, arterio-sclerosis frequently results, and fatty degeneration of the heart occasionally. Amongst the functional disorders attributed to gout are migraine, vertigo, attacks of asthma and angina, muscular cramps, and lumbago. These various conditions, as a reference to other chapters will show, are not exclusively the result of gout, nor always caused by gout when present with it; and their characters, when associated with gout, are not materially different from those they present when due to other antecedents.

Anatomical Changes.—In a joint which has been the subject of gouty inflammation, the cartilage will be found covered with a bright white incrustation, either in patches or more or less completely. If perpendicular sections be made of this, it is seen to be due to a deposit of minute acicular crystals of sodium biurate in the substance of the cartilage. These form in the stratum of cartilage just beneath the surface, leaving at first a thin layer quite healthy, and in later stages the deposit extends irregularly and by small more or less isolated patches into the deeper parts of the cartilage. In advanced cases the cartilage is quite destroyed and eroded down to the bone. Collections of biurate crystals take place also in the other tissues of the joints, for instance, in the ligaments; and in the cutaneous structures, so that the movements of the joints are cnosiderably impaired; and in some cases this is assisted by some of those changes—osteophytes, &c.—which occur so commonly in osteo-arthritis. In bursæ, in tendon sheaths, in the cartilage of the ear, or in the skin of parts not immediately over the joints, the essential change is also the accumulation of biurate crystals. In the gouty kidney minute yellowish-white streaks, which are due to crystals of the same substance, may be seen, especially in the pyramids. Exceptionally, true gouty deposit has been found in other situations—*e.g.*, on the spinal meninges, and on the meninges of the cerebellum.

Diagnosis.—The typical gouty attack occurring at night in the great toe can scarcely be confounded with anything else. It is distinguished from the arthritis of *rheumatic fever* by the dark-red, shiny, tense swelling of one joint, the absence of general sweating, and the slight constitutional disturbance. Later illnesses implicating many joints show a closer resemblance. There is generally a history of many previous invasions of single joints, and less fever or sweating than in rheumatism: but the condition of the joints themselves cannot be relied upon as it can in early attacks. The swelling and redness of the back of the hand in gout may be such as to closely resemble *abscess*, but fluctuation can scarcely be obtained, and the history will mostly protect against errors. *Pyæmia* may be suggested by multiple gout, but rigors would be more severe, and the constitutional disturbance more intense. In all cases the ears should

be carefully examined for tophi, and, if necessary, Garrod's thread experiment may be used as a test.

Prognosis.—Gout once declared is likely to be repeated unless the conditions, dietetic or otherwise, which have led to it are altered. Acquired late in life, and properly treated, it may not materially shorten life; but it tends to granular kidney, atheromatous arteries, and cerebral hæmorrhage. The risk of these sequelæ is more or less in proportion to the frequency of joint attacks, and hereditary gout developed early in life is very likely to be fatal by uræmia or apoplexy before old age is reached.

Nature of Gout.—The prominent feature of gout is the excess of uric acid in the blood and tissues: but the uric acid is not free in the blood, it is always combined, and it does not act directly as a poison. Several views have been expressed as to the cause of the excess, and the way in which it is related to the articular and visceral manifestations of the disease.

Uric acid is derived from the metabolism of nucleo-protein within the body, and from food ingested, especially from foods which, like meat, are rich in purin-bodies. These substances, oxidation products and amino-compounds of purin, $C_5H_4N_4$, by the action of enzymes are oxidisable into uric acid; and uric acid is again by similar action destroyed to form more soluble compounds. It is alleged that uric acid, directly it is formed from nucleo-protein, unites indissolubly with another product of nuclein-metabolism, namely, thyminic acid (nucleotin-phosphoric acid); but that the uric acid provided by the purin-bodies of food has less opportunity of being fixed by thyminic acid, and so tends to increase the quantity of uric acid available for deposition as biurate, if not destroyed by oxidation processes. A synthetic production of uric acid within the body also takes place. It is still open to question whether the excess of uric acid is then due to a deficiency of the enzyme (*oxidase*) which is necessary to destroy uric acid, or to a diminished excretion by the kidney.

In so far as the excessive production or diminished destruction of uric acid is concerned it is regarded by many as highly probable that auto-intoxication, either by the products of faulty metabolism or of imperfect gastro-intestinal digestion, may be operative.

The acute articular inflammations of gout are attributed to irritation by the crystalline deposit of sodium biurate, which is found in the tissues: and possibly the visceral attacks are due to a deposition, at least temporary, of the same biurate crystals. The combination of uric acid with sodium is almost insoluble in blood-serum; it is probably on this account difficult of removal by the kidneys, and at a certain point of accumulation is precipitated in the crystalline form. The selection of particular tissues for the deposition of sodium biurate has been attributed to their low vitality, to their deficient circulation, or to their exposure to injury; and the cartilage of the great-toe joint is regarded as illustrating all these points. Roberts attributes the selection to the richness

of such tissues in sodium salts (synovia, cartilage), and partly to exposure to cold. Others (Ord, Ebstein) have assumed that a local degenerative or necrotic change invites the deposition of the salt.

Treatment.—If this be based on the uric acid theory of gout the objects to be attained in those who are "gouty," or have had attacks of the acute disease, are to prevent the accumulation of urates in the system, and to favour their excretion by the natural channels. These objects can be best effected by judicious diet, by exercise, and, to a certain extent, by some drugs. But it will be seen that a careful regimen, both as to diet and exercise, will also help to obviate the faults assumed on modern views to be the cause of gout. The quantity of food taken should always be moderate. Highly nitrogenous and highly saccharine foods are alike undesirable: but if the relations of purin metabolism to uric acid and gout are established, it is the purin-contents of the food, rather than its richness in nitrogen, which should be considered. The foods which contain no purin are: milk, eggs, butter, cheese, white bread, rice, sago, fruits, cabbage, lettuce, cauliflower. Potatoes contain ·02 grammes per kilogramme, peas ·4, oatmeal ·5, beans ·6, asparagus ·2, codfish ·5, plaice ·7, salmon 1·1, halibut 1, chicken 1·3, mutton ·96, veal 1·10, pork 1·20, ham 1·10, beef 1·10 to 2, liver 2·7, sweetbread 10. Thus white fish and mutton are the most suitable among fish and red meats, and liver and sweetbreads are highly unsuitable. Potatoes should be taken in moderation, on account of the large amount of starch they contain. Personal peculiarities may have to be consulted, so that indigestion is avoided, and with this proviso fat may be taken in moderation. Alcohol in any form is best avoided by those with a tendency to gout; though wines are free from purin-bodies, and, on the other hand, beer, tea, and coffee contain small amounts, the last two as methyl-purins (caffein, thein). A light claret or light dry sherry is the least harmful; or a small quantity of good brandy or whisky well diluted. The stronger sherries, port, burgundy, and champagnes must not be taken. As in diabetes, saccharin may be usefully employed to sweeten beverages such as cocoa or coffee. The advantage of exercise in promoting digestion and assimilation is sufficiently well known. It may be supplemented by the morning bath with the use of the flesh brush or by the occasional use of the Turkish bath with shampooing, or more scientific massage.

The drugs that may be of use in the prevention of gout are chiefly such as aid digestion and promote a free action of the bowels. Occasional saline aperients may be desirable, but free purgation should not be often indulged in. It is common, also, to administer the salts of potassium and lithium, since the urates of these metals are more soluble than the urate of sodium. The citrate of lithium may be given in doses of from 5 to 10 grains, dissolved in water. Thyminic acid, as a drug which combines with and prevents the precipitation of uric acid, has also been given in doses of from 5 to 10 grains three times daily.

Acute Gout.—The affected foot should be kept raised, and supported on a chair or couch, if the patient is not in bed. It should be wrapped in cotton-wool, and in severe cases some anodyne application, such as belladonna liniment, tincture of aconite, tincture of opium, or a lotion of atropine and morphia, may be previously applied sprinkled on lint, and some oiled silk or thin gutta-percha laid over all. Leeches, hot poultices, and ice applications are to be avoided. The diet must be at once restricted in younger patients to milk and farinaceous foods, but in older persons and those broken down by previous attacks it may be more liberal, but still mostly fluid and easily digestible. Alcohol should, if possible, be entirely withheld. Medicinally, colchicum has a decided effect in most cases. It may be given as wine or tincture in doses of 15 to 25 minims every six or four hours, in combination with bicarbonate or citrate of potassium, or citrate of lithium. The bowels should be kept active. If pain is very severe, morphia may be given by subcutaneous injection or internally.

Chronic Gout.—This requires the general treatment sketched for those who have shown gouty tendencies; but as the system becomes more and more broken, depletory and lowering measures must be employed with more caution. Acute outbreaks may be treated with colchicum, and even less active symptoms with the same drug, in smaller doses more continuously. Guaiacum, potassium iodide, benzoic acid, ammonium benzoate, sodium salicylate, and thyminic acid are also often used, and as an adjuvant tonic especially quinine. Alkaline and saline waters are of service, and more still residence at the spas, where the influence of the waters is combined with a regulated diet, fresh air, and pleasant surroundings.

OSTEO-ARTHRITIS

Under this name will be described the disease which has at different times been called *rheumatic gout*, *chronic rheumatic arthritis*, *rheumatoid arthritis*, *arthritis deformans*, and *nodose rheumatism*.

Many believe that two or more different diseases are included under the term osteo-arthritis, and there is sufficient variety among the cases to account for this view. The question whether such differences are due to differences in the age of the patient or his tissues, in the structures primarily involved, or in the nature of the infection, if it is an infection, still awaits solution.

Ætiology.—Osteo-arthritis occurs at all ages, and in all conditions of life; but it is more common after forty years of age than before, in women than in men, and in the poorer classes than in the wealthy.

Cold and damp are popularly regarded as its causes, and they probably predispose to its occurrence, and excite fresh attacks. The antecedent morbid conditions to which most attention has been attracted are previous rheumatic fever, tubercular associations,

sources of possible sepsis such as decayed teeth, pyorrhœa alveolaris, and ovarian and uterine irritation in women. Thus auto-intoxication, either from such sources or from the gastro-intestinal canal, is regarded by many as the probable cause of the disease; but its bacteriology has not made much progress, though organisms have been found in the synovial fluid of the diseased joints.

Morbid Anatomy.—In the early stages there may be effusion into the joint, the synovial membrane is vascular and thickened, and the articular cartilage undergoes softening. This softening begins with proliferation of the cartilage cells, and the matrix splits into fibres perpendicular to the articular surface; then the cartilage cells burst into the joint, and leave a soft ulcerated surface. By a continuation of this process the cartilage is completely destroyed, the bony surfaces come into contact, and the effusion is absorbed. The adjacent surfaces, from continued friction, acquire a dense, hard, white surface (*eburnation*), and may be more or less grooved or fluted on the surface; further, what was formerly a rounded or convex surface becomes flattened down, and considerable atrophy of the head and neck of a bone (*e.g.*, the femur) may in this way take place. At the same time, deposits of cartilage form along the edge of the articular surfaces growing into the capsular ligaments; and these deposits subsequently calcify. They may thus form a kind of ridge or "lip" around the joint, or in some cases (*e.g.*, in the knee) may contribute to produce large plates of calcareous matter, which surround the joint like plates of armour. The synovial membrane also forms large fringes, which may in like manner be invaded by cartilaginous deposits.

Symptoms.—The differences to which reference has been made are chiefly in the age of the patient, the acuteness of the onset, the number of joints involved, and the structures chiefly affected. In most cases it is subacute or chronic, affecting middle-aged persons, involving a few joints, or even one only, subsiding and recurring in these and others, and leading to well-marked osseous changes. In a minority of cases it is acute, multi-articular, in young persons, and largely synovial.

Acute osteo-arthritis and acute rheumatoid arthritis are the names given to the latter form. It occurs especially in young women, begins with acute fusiform swelling of the proximal joints of the fingers, with pain and tenderness, and subsequently the wrists, elbows, shoulders, and joints of the lower extremities are involved, as well as those of the spine and jaw. There is febrile reaction with quick pulse for some weeks; and when this subsides the joints are still limited in their movements, and relapses of fever and joint swelling take place from time to time.

In older persons, the disease may occur with a somewhat rapid onset and affect many joints. Those of the hands and fingers are swollen, painful, tender on manipulation, and stiff on movement. The skin is shiny and slightly reddened. These symptoms subside

and recur from time to time, each recurrence leaving more change behind it; or the disease may be more chronic from the commencement. The stiffness is often most marked in the morning, so that movements are at first painful; yet if they be persevered in, the pain will gradually wear off. Similar symptoms may be noted, in different cases, in other joints, such as the wrist, elbow, and shoulder, the hip, knee, ankle, and foot. The sterno-clavicular articulation, the joint of the jaw, and the vertebral joints may be also involved. Sometimes effusion can be readily recognised, and the joint is tense and elastic. In other joints, or in later stages, creaking or grating can be elicited on passive movement. As time goes on, movement becomes more and more limited from destruction of the articular surfaces and the development of osseous structures in the ligaments and tissues round the joints; and, finally, a fibrous ankylosis may result.

When the disease arises in old people, it is liable to attack one joint, and to cause very extensive changes with destruction of cartilage, and many osteophytes.

Very characteristic deformities take place in consequence of the joint-changes and the muscular atrophy which is associated with them. Thus, the fingers, instead of remaining in line with the metacarpal bones, deviate to the ulnar side, and the joint at the base of the index finger is often greatly swollen: the metacarpo-phalangeal joints are commonly flexed, the first phalangeal joints are over-extended, and the second are flexed. But other forms occur in which these joints are differently placed; and these varieties probably depend on the extent to which the interosseous and other muscles are affected. The lower ends of the radius and ulna project at the back of the wrist. The muscles commonly observed to be atrophied are the interossei in the hand, the muscles at the lower end of the femur, and the deltoid over the shoulder-joint. The nerves also in connection with the joints are affected by chronic neuritis.

In the hip-joint, which is often affected alone, the pain and stiffness are followed by very limited movement, apparent shortening of the limb, flattening of the buttock, and eversion of the limb. The knee and the joint of the jaw are others that may be alone involved.

Heberden's nodes, the small nodules or knobs which may form at the sides of the terminal phalanges, are regarded as an early form of the disorder.

The condition of the joints can be demonstrated by the use of Röntgen rays; and it is often seen that the shadow of the bones is remarkably wanting in density, especially at the articular ends.

In some cases the exacerbations of arthritis are accompanied by constitutional disturbance and pyrexia. But more often in chronic cases fever is entirely absent, and the illness is characterised by anæmia, weakness, and indifferent appetite. There is also in some cases excessive pigmentation of the skin irregularly distributed.

In children a multiple osteo-arthritis is often associated with enlargement of the lymphatic glands and spleen, anæmia, sweating, varying pyrexia, and arrest of the bodily development (Still).

There is considerable variability in the local symptoms—they may subside for long periods, even when untreated, and then burst out again ; they can be relieved by treatment, but complete cure is uncommon ; and a repetition of the complaint is almost certain to occur at some time or other. In most cases there is no cardiac complication, but endocardial murmurs are occasionally heard. The disease is not in itself fatal, but may co-exist with Bright's disease in older people.

Diagnosis.—Chronic *gout* is generally distinguished from osteo-arthritis by the history of the first acute attacks, and the presence of urate deposits (tophi) in the ears and in the affected joints ; if necessary, the blood may be examined for uric acid. As compared with *chronic rheumatism,* we may be confident we are dealing with osteo-arthritis, if the joint-changes are extensive with signs of erosion of cartilage, and growth of bone, or if the disease occurs in a young person independently of preceding rheumatic fever. In the acute cases other forms of acute or subacute poly-arthritis must not be forgotten, such as congenital syphilitic arthritis and gonorrhœal arthritis : the localisation is often different, and the history will help. Arthritis affecting one joint only may be confounded with more surgical forms of lesion. The joint-changes accompanying *tabes dorsalis* generally begin with an abundant painless effusion into the joints, and there is subsequently great mobility, with destructive changes. They may be distinguished by these features or by other evidence of tabes dorsalis—namely, Argyll-Robertson pupil and loss of knee-jerk.

Treatment.—Though but little is known as to the actual cause of osteo-arthritis, it is reasonable to remove such conditions of ill-health as experience has shown to be open to suspicion. Thus the teeth should be examined, and pyorrhœa alveolaris should be treated ; in women, leucorrhœa and pelvic discharges should be dealt with. The state of the bowels should be considered and constipation should be relieved. When there is good reason to suppose that the intestinal contents are unhealthy, if the motions are unduly offensive and fermenting, treatment may be directed to the bowel ; thus, intestinal antiseptics have been given, such as salol, bismuth salicylate, and β-naphthol ; or the colon has been frequently irrigated. The "soured milk" treatment may be also tried. This consists in the administration daily of from one to two pints of milk containing cultivations of the lactic acid bacillus, preferably the bacillus of Massol or the Bulgarian bacillus, on the hypothesis that this bacillus in the alimentary canal will drive out all other more harmful organisms. Unfortunately those methods based on imperfect knowledge of the pathology of the complaint are not always successful. Still's disease is more obviously than other forms due to a general infection ; and much good can be done with arsenic.

In the treatment of osteo-arthritis in its usual forms the diet should be carefully ordered so as to avoid indigestible articles, but should not be stinted; indeed, the patients require, as a rule, good feeding, and meat may be given freely, as well as vegetables. A moderate use of alcoholic drinks may be allowed. The patient should be well clothed in flannel, a warm dry atmosphere should be looked for, and changes of temperature avoided. Various health resorts and spas fulfilling these requirements have been found beneficial, such as Buxton, Bath, Harrogate, and Strathpeffer, at home, and Aix-les-Bains, Aix-la-Chapelle, Baden-Baden, and Wiesbaden, abroad. Internally, arsenic, aspirin, sodium salicylate, guaiacol, iodide of iron, and cod-liver oil are most valuable, and they must be continued, with such intermissions as may be desirable, for weeks or months. The arsenic should be given in full doses.

Locally much benefit may be derived from the application of tincture of iodine, small blisters frequently repeated, frictions with stimulating liniments, and passive movements, burying the joint in hot sand, dusting it with flowers of sulphur, and wrapping it in flannels, the continuous current daily applied, electric light baths, hot-air baths, and high-frequency currents.

After pain has subsided, massage will help to restore the mobility of the joints and the muscular nutrition.

Any method that is adopted must be persevered with for some weeks before being given up as of no value.

CHRONIC RHEUMATISM

This condition is sometimes the sequel of acute rheumatism, but at others it arises independently in persons of middle or advanced age. There are many who think it cannot be separated from osteo-arthritis, and in our ignorance of the nature of either disease, the question must be left open.

Symptoms.—One or more joints become painful and stiff, and the condition varies much: liable to be worse in the morning, and to improve as the day goes on and the joints are more moved; aggravated by cold and wet, therefore worse in the winter, and improved by the opposite conditions. The joints are tender, but they may show very little change otherwise, or there may be slight swelling, a little heat locally, and perhaps crackling on movement. The general health is not much affected unless the pain is severe and continuous. After a time there may be deformities from muscular action like those seen in gout or osteo-arthritis; and they may become permanent from secondary fibroid changes.

Morbid Anatomy.—As a rule, the joint-changes are slight; there is very little effusion, but some thickening of the synovial membrane and cartilage, and later on, of the capsules and adjacent tissues.

Treatment.—The affected joints should be kept warm; and the pain is often relieved by local counter-irritation, as by painting with iodine, or friction with camphor and other stimulating liniments.

Internally, the salicylates may be tried, but cannot be relied on as they can in rheumatic fever. Potassium iodide, alkalies, and guaiacum may be employed, while the general health and the digestion should be cared for. Relief may often be obtained at the baths of Buxton, Bath, Harrogate, Droitwich, and the Continental spas mentioned under Osteo-arthritis.

RICKETS

(*Rhachitis*)

Rickets is a disease involving the nutrition of infants and young children, of which the chief result is that the bones become enlarged at their epiphysial ends, and so soft that they are bent by the pressure to which they are put in the ordinary use of the limbs.

Ætiology.—It is essentially a disease of children, the majority of cases commencing between the ages of six months and twelve months, but some as early as three months and others as late as two or two-and-a-half years. It is not hereditary. It affects the sexes about equally. The children of the poor are much more frequently affected with it than those of the richer classes, though the latter are not exempt. This is no doubt related to what is regarded by common consent as the chief element of causation—namely, defective hygienic conditions, especially in the matter of food and air supply. The natural food of the infant is the mother's milk, and a child should be nursed entirely until it is nine months old. The faults of diet from which infants suffer are—(1) defective quality of the milk from ill-health or malnutrition of the mother, or from lactation being continued far into the second year; (2) the substitution for the mother's milk of various "infants' foods," of which the larger number contain a high percentage of starch, so that there is a proportionate diminution of the very essential fatty and proteid elements; (3) the ingestion, through carelessness or ignorance of the parents, of meat, bread, and potatoes, or other adult foods, either alone, or in addition to the mother's milk or the artificial substitutes. Overcrowding in close unventilated rooms, and confinement to the house, may also operate deleteriously in the growth of infants. There can be very little doubt that in these several deficiencies lies the cause of rickets in the great majority of cases, and though they may all have some share in it, the results of treatment are in favour of its being largely due to insufficiency of fat, and perhaps of proteids. It does not seem impossible that a defective supply from the mother to the fœtus *in utero* might start the disease even before birth. It is possible also that even if the fatty elements of food are adequately supplied, they may be in some cases imperfectly assimilated.

Symptoms.—Early in the complaint two symptoms occur, which are very constant. One is that the child is restless at night, kicks off its clothes, and lies with its legs and arms exposed. The other is that when it goes to sleep it perspires profusely about the head and neck, so that the pillow is saturated. But the rest of the body is often dry, and the temperature is normal. The first evidence of changes in the bones is seen in the *enlargement* of the *epiphysial* ends of the long bones. This is well marked at the wrists, where the ends of the radius and ulna are thickened, and at the ankles and at the knees; but it is perhaps most unmistakable at the junctions of the ribs with the costal cartilages, where a series of nodules are formed, reaching on either side from the first rib near the sternum downwards and then outwards to the twelfth rib in the flank. This has been called *beading* of the ribs, or the *rickety rosary*. The defects of ossification are seen in the skull, where the fontanelles are large, and may not close until long after the usual time, which may be put at about eighteen months. Another symptom is the *delay in the eruption of the teeth*, the first of which may not appear until the eleventh or twelfth month, instead of the sixth or seventh; and the order of their appearance may present many irregularities.

Accompanying the enlargement of the ends of the bones there is an abnormal softness, in consequence of which the bones yield to the traction of the muscles or the weight of the child's body, and become bent so as to produce characteristic deformities of the limbs, the chest, and the pelvis; the head also acquires a peculiar shape, though it is less easy to see how this happens. If the disease comes on after the child has begun to stand or walk, these accomplishments are given up, and the child "is taken off its feet," as the mothers are apt to explain. If the disease begins earlier, then the art of walking may not be attained until the eighteenth or twenty-fourth month. In either case the child tries to walk before the bones are completely consolidated, and the weight of the body causes the tibiæ and femora to be bent or "bowed," generally with a convexity outwards and forwards. Sometimes there is a convexity inwards at the lower part of the tibiæ, the feet being thus widely separated, and this is to be attributed to the child getting about the floor in a sprawling position, using the feet like the hind fins of the sea-lion. If, while still unable to stand, the child crawls much about the floor, the weight of the body falls upon the arms, and the radius, ulna, and humerus get correspondingly bent. In the chest, deformity is produced by the action of the diaphragm, which sucks in the ribs at the softest part; thus is produced a wide groove on either side of the sternum. The sternum is prominent, and the upper part of the chest has a somewhat square shape; the lower ribs, however, are often expanded over the abdominal viscera, forming the upper arch of a tumid abdomen, which contrasts strikingly with the narrow chest above, and is separated from it by a transverse depression (Harrison's sulcus). The pelvis does not usually show any deformity in infancy,

but in extreme cases of rickets the pelvic aperture is considerably misshapen, being mostly of an hour-glass type, and it may afterwards, in females, offer very serious obstruction to parturition.

The head, besides presenting large fontanelles and often lines of depression corresponding to the coronal and sagittal sutures, acquires a somewhat square shape, the vertex being flattened, and the frontal and lateral regions being rather prominent. In pronounced cases the cranium looks very large in proportion to the face, but contradictory statements are made as to its actual size; for while some say that hypertrophy of the brain and distension of the ventricles (hydrocephalus) are common accompaniments of rickets, it is stated by others that the head is not really enlarged, but only seems so because the facial bones are ill-developed and stunted in growth. I believe that the circumference of the skull is often abnormally large, but it does not therefore follow that the contents are also greater; since the contents of a cube are less than those of a sphere of the same superficies.

In extreme cases there is considerable stunting of all the bones, as well as the shortening by curvature; and children of eight or ten may be no taller than those of three years old. The bones are also more fragile than normal, and *green-stick fractures* are apt to occur. There is decided atony of the ligaments of the joints, and of the muscles: the latter is shown in the prominence of the abdomen. The liver and spleen are easily felt because they are pushed down by the contracted chest, and are ill-supported by the abdominal muscles. The appetite may be very good, and many rickety children show a perfect or even excessive development of fat, but in severe cases there is anæmia; if any disturbance of the stomach or bowels is present it is to be attributed to diet, which may have caused the rickets, and not to the rickets itself. On the other hand, the nervous system is seriously involved; rhachitic children are very liable to infantile convulsions, including the special forms known as *laryngismus stridulus* (*see* p. 475) and *tetany*. Associated with laryngismus is often *facial irritability*, or Chvostek's sign, in which a gentle tapping over the superficial branches of the motor nerves of the face causes contraction of the corresponding muscles. In rickety children also, rather more frequently than in others, occurs the disorder known as *head-nodding* or *head-shaking* (*spasmus nutans*), which is often associated with *nystagmus*. These occur in babies of from four months to one year; they are most frequent in the dark months of the year, December and January, and in crowded localities, and they subside as the child grows older.

Rickets is essentially a recoverable disease in the sense that it does not directly cause death, and that the process of bone-softening ceases after a time, although it may have produced deformities which are permanent. If the disease is but slight it may leave no traces in after-life, and this is probably the case with the majority of patients: the bones become hardened, and the limbs ultimately

become perfectly straight. But in other cases the effects of former rickets may be seen in the big square head with prominent forehead, the curved femora and tibiæ, and the pigeon breast of the adult. Next to fits and laryngismus the most serious result of rickets in childhood is the aggravation of bronchitic attacks which the soft state of the ribs causes. By their want of rigidity the act of coughing especially is rendered imperfect, and the secretions accumulate, to the imminent danger of the child. Death is often brought about thus: and in other cases the frequent occurrence of bronchitis, by the collapse of lung which it produces, helps in the formation of the pigeon-breast.

Morbid Anatomy.—The changes in rickets are best seen at the ends of the long bones, or of the ribs. If the swollen portion at the junction of the rib and its cartilage be divided longitudinally, it will be seen that the line between the two structures is remarkably irregular, instead of being quite straight, as it is in healthy bones. Normally, between the already developed bone and the unossified cartilage are two narrow bands, one bluish-grey—the zone of proliferation; the other of a yellow colour—the zone of ossification. These are very narrow, quite straight, and parallel. In rickets the proliferating zone is thickened, reddened by new vessels, and has thrown out processes irregularly into the cartilage and bone on either side. Under the microscope it is seen that the proliferation of cartilage-cells, preparatory to ossification, has taken place with great freedom, but with no uniformity as it does in health; that calcification has begun unduly early in some cartilage-cells, whereas it is deficient in the trabeculæ of cartilage. The processes of proliferation of cartilage-cells, of deposition of calcium-salts, and of formation of medullary spaces, take place not in a uniform, regular, or progressive way, but in a most disorderly manner, and with varying degrees of rapidity at different spots. Analogous changes are seen on the surface of the bone where it is formed from periosteum; here is a soft vascular layer much thicker than is normal, showing a similar activity of the early stages of transformation, and delay in the deposition of calcareous salts. The whole bone also is unusually vascular, and the contents of the medullary cavities are redder than normal.

The muscles are flabby and wasted. The blood in anæmic cases shows a great deficiency of red blood corpuscles, a proportionate or greater fall of hæmoglobin, some leucocytosis, and the presence of red nucleated cells, especially normoblasts. The changes in the spleen, liver, and glands, when they occur, appear to be due to increase of interstitial connective tissue.

Pathology.—With regard to the nature of the disease, it cannot be said at present that anything is really known of the link between the defective hygienic conditions and the bone-changes and general symptoms which result.

Diagnosis.—This rarely presents difficulties. The important early signs are the sweating of the head and the dislike to being

covered at night, the beading of the ribs and the thickening of the wrists, and the apparent enlargement and shape of the head. Sometimes the inability to walk may lead to a suspicion of infantile paralysis, but the limbs can at least be moved, and the deformities of the bones should give the right clue.

Prognosis.—Recovery is the rule, the bones ultimately becoming quite firm and solid; but the deformities, if considerable, will be perpetuated. The risk to life is from the complications, especially bronchitis, with collapse of lung, convulsions, and laryngismus stridulus.

Treatment.—The first essential is the improvement of the food and general hygiene of the child. It should live in well-ventilated rooms and should be taken out in the fresh air regularly. It should be warmly and suitably, but not too thickly, clothed. If it is being nursed, it must be understood that the supply of food is good and abundant. This is not likely to be the case if the mother is delicate, or if the nursing has been continued into the second year. In the former case additional food—*e.g.*, cow's milk, diluted with one-half, one-third, or a less proportion of water, according to the age of the child—should be given; in the latter, the nursing should entirely cease. Starchy foods, for the digestion of which the infant's secretions are by no means prepared, should be excluded, and it is best to let a good cow's milk form the chief element in the diet, to which lime-water and a little cream may be added; indeed, the directions given in a former chapter should be followed (*see* p. 729). As the infant approaches the end of the first year, beef-juice, chicken-broth, or gravy may be added; and at a later age, well-boiled cauliflower, a little pounded mutton, the yolk of a boiled egg, or some custard pudding. Milk should still form a large part of the child's diet, and starchy foods should be given sparingly, if at all. The most valuable medicine is no doubt cod-liver oil, which should be given two or three times daily after a meal. The dose may be fifteen drops for an infant six months old, twenty drops up to twelve months, half a drachm up to eighteen months or two years, and a drachm for older children. Iron is often employed, as syrup of the phosphate; and preparations of calcium, such as the lacto-phosphate, are recommended, although it will have been seen that the disease is a good deal more than a mere deficiency of calcium-salts in the bones. Phosphorus has been much used abroad in doses of $\frac{1}{120}$ grain once or twice daily, but it is generally given dissolved in oil, which may have some share in the result. When the child is under treatment, and the bones are soft, it is desirable to prevent their being bent by the weight of the child's body. Walking should be forbidden, and it may be conveniently prevented by fixing to each leg a flat splint, projecting three or four inches beyond the foot; these may be removed at night. The deformities of the limbs which remain after rickets is cured may, if extreme, be treated surgically.

FŒTAL RICKETS AND LATE RICKETS

The relations to rhachitis of cases denominated fœtal and late rickets have yet to be cleared up. It would seem that the former has some resemblances to cretinism; and cases of the latter class, in which at the age of eight or ten years the bones become soft and deformities arise like those of true rickets, may prove to be divisible into two or more groups—one, perhaps, a true rhachitis, another more like the osteomalacia of adults.

ACHONDROPLASIA

This is a congenital disease of the bones and cartilages, which results in permanent stunting of growth and other deformities. It has no doubt been often mistaken for rickets; which at the same time may sometimes complicate it. At birth the limbs, especially in their upper halves, are noticed to be abnormally short. The child may be of full weight, but growth is slow, the limbs continue to be short, and the stature is consequently small, though the vertebral column is of normal length. There is a projection of the buttocks which gives the appearance of lordosis. The head is generally large, somewhat like a rhachitic head, with a prominent forehead, and depressed bridge of the nose. The hands present a characteristic deformity in that the index and middle fingers diverge from the ring and little fingers when the hand is open. The deformities are due to defects in ossification of the cartilages of the long bones, of the ribs, and of the innominate bones, together with a premature synostosis of the bones of the basis cranii. The cranial bones which are formed in membrane are normally developed, as well as the bones which remain cartilaginous till a late period of fœtal life, viz., the sternum, patella, carpal and tarsal bones; and the costal cartilages. The thyroid is normal; and the subjects of the disease, who survive to adult life, have a good or average intelligence. No treatment is of any avail.

ACROMEGALY

In this curious disease, first fully described by Marie in 1880, there is an enlargement of the bones of the extremities and of the face (*ἄκρον*, an extremity, *μέγας*, large).

Ætiology.—It begins in adolescence, or early adult life, and affects women somewhat more often than men; beyond this no causative conditions have as yet been traced.

Symptoms.—It is a very chronic disease, and the early stages are accompanied by pains in the head or limbs, by languid feelings and weakness, and in women by cessation of the menses. Then it

is observed that the hands, fingers, nose, and face are becoming gradually larger. In an advanced condition, the nose is long and thickened, the lower jaw is enlarged transversely, and projects beyond the upper jaw, while the teeth are widely separated. Occasionally the upper jaw is also affected. The skin of the face is coarse and thickened; this is well seen in the eyebrows and the soft parts of the nose; the lobe of the ear is hypertrophied, the tongue is large, and the voice becomes deeper. The hands and fingers are much enlarged, the hands broad, the fingers thick and like sausages, and the nails coarse, broad, flat, and ribbed; but the wrist is generally small. The feet are involved like the hands. Other bones may be also affected, and there is some bending of the back (kyphosis) and consequently diminution of stature; but the long bones are not generally bent.

Associated with these changes in the bones and integuments are some suggestive internal lesions. The thyroid gland is sometimes atrophied, at others hypertrophied; the thymus gland may be persistent and enlarged, and may cause dulness at the manubrium sterni; and *post mortem* the pituitary body has been found to present hyperplasia of its follicles with sclerosis of the vessels and other tissues, or to be the subject of tumour. There is in some cases double temporal hemianopia, or varying degrees of optic atrophy, or even complete blindness. The cerebral functions may be affected: thus loss of memory, slowness of cerebration, depression, delusions, unstable temper, and somnolence are frequent; and fits occasionally occur, glycosuria is frequent, and nephritis with albuminuria is a complication in many cases. The disease lasts several years, but no improvement takes place.

Diagnosis.—Acromegaly must be distinguished from giant growth, or *gigantism*, and from *leontiasis ossea*. The former is noticed at birth, or begins in childhood, and the enlargement is nearly uniform, or at most the limbs are disproportionately long. These overgrown individuals are rarely long-lived. In leontiasis only the bones of the head and face, especially the jaws and malar bones, are hypertrophied, and the tumour-like enlargement is often unsymmetrical.

Pathology.—The disease is no doubt closely related to disease of the pituitary body, but whether this operates by defect of or by increase of its internal secretion cannot be certainly said. Moreover, other organs with internal secretions, such as the thyroid, ovaries, and testes, are often affected at the same time.

Treatment.—The various animal extracts of the thyroid, thymus, and pituitary body are being tried, but not much success has been recorded.

HYPERTROPHIC PULMONARY OSTEO-ARTHROPATHY

This is a condition of enlargement of the bones, which is most obvious in the hands and feet, and occurs especially in cases of chronic pulmonary disease, such as phthisis, empyema, bronchiectasis, and cancer, but also in some diseases of the liver, such as hypertrophic cirrhosis (*see* p. 779) : and the clubbed fingers of congenital heart disease (*see* p. 612) appear to be indistinguishable from it. The fingers are longer and thicker than normal, and the nails are striated, fibrous, and curved over the much-clubbed ungual phalanges. The carpus and metacarpus are less affected. The feet and toes are similarly altered, the ends of the long bones may be thickened, and there may be effusion into the joints, preceded by stiffness. Pathological and skiagraphic observations show that the essential change is a thin deposit of new bone under the periosteum of the long bones, and especially of the lower thirds of the radius, ulna, tibia, fibula, metacarpals, metatarsals, and proximal and middle phalanges of the hands and feet. The deposit is such as to fill up the concavity between the proximal and distal ends of the bone, and to make, for instance in the case of a metacarpal, the middle thicker than the ends. Sometimes the joints (wrists, ankles, and interphalangeal joints) contain fluid, and the synovial membrane is thickened. The thickening or clubbing of the fingers and toes is due to excess of fat and fibre in the soft parts.

The condition is usually progressive, but improvement has occurred coincidently with relief of the causative disease.

The generally accepted explanation is that it is due to absorption of toxins from septic organisms, such as are present in phthisis, empyema, and bronchiectasis. In hepatic cirrhosis, the liver probably fails to destroy toxins proceeding from the alimentary canal : and in many cases of heart disease there is an associated pulmonary lesion which may provide the poison.

MOLLITIES OSSIUM

(*Osteomalacia*)

In mollities ossium bones which have developed normally become soft and fragile.

Ætiology.—It is a disease of adults, beginning mostly between the ages of thirty and forty ; and it affects women almost exclusively. Little more is known of its ætiology, except that it often begins during pregnancy, and is aggravated by it ; and that it has been much more often observed in some parts of the world (along the Rhine, Westphalia, Switzerland near Bâle, North Italy, and Calabria) than in others.

Symptoms.—The first is pain, which begins in the lower half

of the spine, the pelvis and the loins; or in the feet, knees, or other parts of the lower extremities. It varies much in severity and persistency, and may wander from part to part, or remain fixed in one spot for a long time. With this the patient becomes languid, and is disinclined to do anything. In course of time a change of figure may be noticed, the patient loses height from rounding of the spine or bending of the limbs: then, some day or other, a bone breaks, with very little apparent cause, or on slight exertion. As a rule, the fracture heals but imperfectly; and subsequently other bones break. Moreover, the long bones show a remarkable degree of flexibility, so that they can be bent into very strange positions, and the more superficial bones may be indented with the finger. Though the general health may be preserved at first, exhaustion at length supervenes.

The disease runs a chronic course, and generally lasts from four to six years. Death takes place often from inability to breathe on account of the softness of the ribs; sometimes during parturition from the obstruction which the distorted (rostrate) pelvis offers to the passage of the fœtus.

Morbid Anatomy.—The bones are found to be so soft that they can be cut with a knife or indented with the finger. Nearly the whole of the bone is converted into a soft greasy mass or pulpy material, except, perhaps, a thin shell of compact tissue just under the periosteum; or even there may be nothing left but the thickened periosteum itself. Microscopically, the change seems to be, first, a confusion of the natural minute structure of the bone; the Haversian systems become fused together, and then absorption of the salts takes place from the innermost rings round a canal, so that the substance is gelatinous and transparent. As the same change proceeds in the rings placed farther out, the innermost rings are entirely absorbed, the lacunæ also alter their shape and size, and finally the Haversian systems are destroyed. The bone becomes spongy and porous, and the enlarged medullary spaces are filled with a marrow which is fatty, or lymphoid, with giant cells and osteoblasts. In this there are recent hæmorrhages and pigment resulting from former effusions of blood. In some places, the medulla may be gelatinous, or watery. Chemical analysis of the bones shows that the inorganic constituents are reduced from 68 to 30 per cent.

The change in the bones was formerly attributed to the presence of lactic, carbonic, or other acid in the blood; but such theories have been abandoned. Some associations of osteomalacia and goître have been observed; and in some cases hyperplasia of the parathyroids followed by atrophy and small-celled infiltration has been found. The intimate connection with the ovaries suggested by its occurrence and aggravation during pregnancy is confirmed by the fact that the disease has been arrested by the removal of these organs; but this hypothesis will not explain infantile, senile, or male cases.

The **Diagnosis** is not difficult when the changes in the bones

have become manifest. Earlier symptoms may be mistaken for *rheumatism.* Spontaneous fracture also may take place in *sarcoma, carcinoma,* and *multiple myeloma* of the bones.

Treatment.—Porro's operation and the removal of the ovaries have been advocated, and several times performed; the existing deformities remain, but further progress is checked. Otherwise, recovery is exceedingly rare. The treatment should be tonic, as by quinine, iron, and cod-liver oil. Foods containing lime and phosphorus should be given, such as milk, eggs, fish, fresh meat, peas, beans and cereals; and phosphorus is said to be of value in doses of $\frac{1}{20}$ to $\frac{1}{12}$ grain daily, either as oleum phosphoratum or pil. phosphori (B.P.), continued for one or two months.

MYELOPATHIC ALBUMOSURIA

The above name is given by Dr. T. R. Bradshaw to a disease of the marrow of bones, with which is associated the presence of Bence Jones' albumose in the urine (*see* p. 904). The disease has probably in the past been confounded with mollities ossium. It occurs more often in males than in females, and as a rule in the second period of life—that is, after thirty-five years of age. The change in the bones is a new growth commencing in the interior of the bone, and sometimes coming through the compact tissue, so as to form a considerable tumour. The growth is gelatinous, soft and vascular, and has a general resemblance to red marrow, being composed chiefly of round cells, with little or no intercellular substance. Many growths occur on the same patient, and the bones usually involved are the ribs, sternum, and vertebræ, less frequently the pelvis and skull, and rarely the long bones of the limbs. These tumours have been described as *multiple myelomata.* The disease comes on gradually, and the condition of the bones may give rise to the first symptoms, or the urine may be accidentally found to contain albumose. In the former case there are pains in the back and sides, increased by movement, accompanied by tenderness, and varying in position from day to day. Tumours are sometimes felt on the ribs, and the dorsal or lumbar vertebræ may be deformed; in many cases spontaneous fractures have occurred. There are anæmia and increasing weakness, and death takes place in from one to three or four (exceptionally eight) years from the first symptom. No treatment is known for it.

Multiple myeloma may exist without albumosuria; but it is doubtful if Bence Jones' albumose ever appears in the urine apart from the disease of the bones.

DISEASES OF THE SKIN

The skin is liable to the same pathological conditions as other organs and structures in the body, and the classification of its diseases is based upon these.

Thus there are changes in vascularity, inflammations, new growths, structural changes due to the presence of micro-organisms, such as those of tubercle or leprosy, lesions resulting from the circulation of disease-toxins, such as those of scarlatina, syphilis, small-pox, and other exanthemata, the invasion of animal and vegetable parasites other than bacteria, hypertrophy or atrophy of the separate structures of the skin, and changes of pigment.

We have to consider also the disorders of certain organs contained in the skin, namely, the hair, the sebaceous glands, and the sweat or coil glands.

Many disorders of the skin have been already described, as, for instance, the eruptions characteristic of the exanthems, the cutaneous lesions of syphilis, lepra, glanders, actinomycosis, and elephantiasis, and the forms of hæmorrhage known as purpura.

The discrimination of the various diseases is based upon a careful observation of the local changes which take place in the skin, combined with a consideration of their distribution, duration, and associated and antecedent circumstances.

These local changes are known as *elementary* or *primary lesions.*

Hyperæmia.—This consists of redness, of varying extent, due to the blood-vessels being distended with blood. It may be—(1) an *active hyperæmia* from vasomotor paralysis; (2) an *early inflammatory hyperæmia,* with slight swelling, tenderness, and some indications of pyrexia; or (3) a *venous hyperæmia,* or passive congestion, with a more blue or livid colour than in the other forms. In all cases the redness disappears on pressure, to return when the pressure is removed—quickly in the first two cases, slowly in the last.

Hæmorrhages.—Cutaneous hæmorrhages form larger or smaller spots of bright red, dark red, or purple colour: they do not disappear on pressure. As a rule, the blood is gradually absorbed, and the colour fades into brown or brownish-yellow, or becomes successively brown, green, and yellow, in the large subcutaneous hæmorrhages. A yellowish-brown stain may be left for a long time. The smaller spots are called *petechiæ,* the larger *ecchymoses:* if they form streaks they are called *vibices.* In *capillary ecchymosis* a very fine mottling is produced, which looks like a hyperæmia, until it is found to persist under pressure. Exceptionally the skin involved in hæmorrhage sloughs and leaves an ulcer.

Papules or **Pimples.**—Small red or pink elevations of the skin, solid, or at least not visibly containing fluid. They arise mostly in the cutis, but may be imitated by accumulations of epidermic scales.

Vesicles or **Vesiculæ.**—Small blisters, from 1 to 5 mm. in diameter, due to the accumulation of clear fluid under the upper layer of the epidermis. They are frequently inflammatory, seated upon an inflamed base, and contain a yellow albuminous serum.

Blebs or **Bullæ.**—Large vesicles, from 5 mm. to an inch or more in diameter. The fluid contents are clear, or slightly turbid, or blood-stained. They are often situate on an inflamed base: they heal by discharge of the contents, and the drying and shedding of the epidermic scale.

Pustules or **Pustulæ.**—Vesicles or bullæ containing pus.

Scabs or **Crusts.**—Irregular flat masses of dried serum, pus, or blood, or a mixture of these materials, forming upon and adherent to the raw surface which has secreted them, and frequently the result of a vesicle, pustule, or bulla.

Wheals or **Pomphi.**—A circumscribed œdema of the corium, producing a pale pink or white elevation of the skin.

Scales or **Squamæ.**—Collections of epidermic cells in the form of flakes. Sometimes, as in seborrhœa, there is a large admixture of the fatty matter of sebum. Scales vary from the small branny particles of measles (furfuraceous) to the large exfoliations of exfoliative dermatitis and some cases of scarlatina, or the thick adherent masses of psoriasis.

Scratch Marks.—Linear lesions of the skin, from a third to two inches in length, produced by the nails, and bearing small crusts of blood. Ultimately, if deep enough, they become linear or fusiform cicatrices. Their direction generally bears a definite relation to the position of one or other hand, and parts of the body which the hands cannot reach are exempt from them.

Raw or **Excoriation.**—A patch of skin deprived of the upper layer of the epidermis, and exposing the stratum mucosum. It is of a vivid red colour, and tender to the touch; and it secretes a small amount of serum, which may dry into a crust.

Chaps, Rhagades, or **Rimæ.**—Cracks or fissures through the epidermis, reaching the stratum mucosum or corium beneath, very sore and apt to bleed.

Sore or **Ulcer.**—A loss of substance involving the epidermis, and extending to the papillary layer. The base is covered with granulations, and secretes pus. It heals by scarring.

Scar or **Cicatrix.**—The new growth of connective tissue, which results from the healing of sores, involving the papillary layer and deeper corium. When recent, they are pink or bluish in colour. Finally they become dead white, and contract in size.

Nodules.—Solid elevations larger than papules. They have been called tubercles—a name which is now best limited to the specific lesion which is the first change in phthisis.

Stains or **Maculæ.**—Small areas of pigmentation deeper than that which is normal to the part. They may arise from a preceding hyperæmia, and then disappear shortly. They are generally more permanent if arising independently.

INFLAMMATORY CONDITIONS, OR FORMS OF DERMATITIS

ERYTHEMA

Erythema (from *ἐρύθημα*, a blush) has a rather wide signification. It includes the redness of the skin which may be set up by external irritants, and the rashes of some infectious diseases. In both these cases there is an early phase of inflammation. A more severe lesion has been called by Hebra *Erythema exudativum*, and is characterised by much more definite and pronounced inflammatory lesions. The skin is red, swollen, and tender; in some cases bullæ or vesicles are formed; and blood may be effused into them or into the corium.

Ætiology.—Patches of erythema and erythematous rashes are produced by a number of toxic conditions, such as bacterial invasion, the circulation of bacterial toxins, the presence of protozoa in the blood (trypanosomiasis), visceral disorders probably producing poisons or toxins from the food, the ingestion of certain drugs and poisonous substances, and the injection of antitoxic sera.

Pathology.—The essential change in erythema is an inflammation of the corium, with dilatation of vessels and exudation of lymph and leucocytes. In different cases the process seems to be more intense at different levels in the skin structures; sometimes the skin alone is swollen, at others the epidermis is raised into blisters. The vesicles may contain either serum, pus, or blood; and the blood extravasated may be a visible hæmorrhage, or only enough to produce staining as recovery takes place.

ERYTHEMA MULTIFORME

As the name suggests, there is a very great variety in the lesions produced. In all cases they are bright or dark-red elevations of the skin, which may be in the form of papules (*E. papulatum*), or in larger patches or nodules, the size of a sixpenny or shilling piece. Such a patch may clear at the centre, and leave a ring (*E. annulatum*); if this enlarges it may coalesce with neighbouring rings and produce sinuous or scalloped patches (*E. gyratum*). *Erythema marginatum* has a similar outline: the peripheral margin of the red band forming the ring is raised abruptly, and the central margin gradually slopes towards the skin. Sometimes a ring of erythema is surrounded

by another ring outside it, and this by another farther out, while the first ring is beginning to fade. The different colours of the rings in various stages suggest the name *E. iris*. I have seen four such rings at the same time.

Occasionally bullæ or vesicles appear on these patches, and sometimes petechiæ or ecchymoses may occupy the centre of a broad papule (*purpura urticans*). The raised patches last a few days and then gradually subside, often leaving a brown or brownish-yellow stain, even if there has been no obvious hæmorrhage into the structure of the skin. The whole duration is from two to four or six weeks. It may begin with some malaise; it is occasionally accompanied by not very definite joint-pains; and it is a not infrequent occurrence to have an eruption of erythema, especially E. marginatum and hæmorrhagic varieties, in the course of ordinary acute rheumatism. The hæmorrhagic forms are called *Peliosis rheumatica*, but hæmorrhages (purpura) certainly occur also in rheumatism without any preceding true erythema (*see* p. 171).

Erythema multiforme occurs more often on the back of the hand and forearm, the front of the leg, and the dorsum of the foot; and on the face, neck, front of the chest, and abdomen.

The vesicular and bullous phases of erythema are comparatively uncommon (*E. bullosum, Herpes iris*). After some preliminary tingling, a small papule forms, which soon shows a minute vesicle upon it. The vesicle enlarges, becomes flat, and is surrounded by a pink areola. After a time the fluid is absorbed from the centre, leaving a purplish depression surrounded by the still vesicular periphery. Or the centre remains fluid, then comes a zone of purplish depression from absorption, then a peripheral zone still fluid, then the areola outside all. The recovery of any patch takes place in about a fortnight by fading of the areola, absorption of the fluid, and subsidence of the papule; but from repeated crops the whole disease may last from four to six weeks. The backs of the hands and fingers, especially the radial half, and the insteps and knees, are the parts most affected; and the disease is generally symmetrical.

In another variety there is a central bulla, and round this a ring of vesicles of smaller size. A second ring may form round the first, and a third round that. In some cases of vesicular and bullous erythema, the contents of the vesicles may be purulent, or sanguineous, and the process may extend deeply enough into the corium to produce ulcers which are followed by scars.

Treatment.—There is no specific treatment for the erythemata as such. Their cause, if it can be recognised, should be dealt with, and relief to discomfort about the lesions can be given by local means. In cases having a rheumatic orgin, salicylate of sodium should be given in doses of 15 or 20 grains three or more times in the day. If gastric troubles have caused the erythema, unsuitable articles of food should be withdrawn, such as shell-fish, salted fish, pork, and sweets; and the dyspepsia should be properly treated. Locally, astringent and sedative lotions are of value, especially lead lotion

(liq. plumbi subacet. dil.), combined with opium if there is much irritation, or calamine lotion or evaporating lotion of spirit and water, eau de Cologne, or liq. ammon. acetatis; in the drying or scabbing stages of the vesicular forms, zinc or zinc and lead ointment. Erythema iris has been successfully treated by the application of a 1 per cent. solution of picric acid on wool (A. P. Allan).

ERYTHEMA NODOSUM

This consists of oval or circular solid flat elevations of the skin, from half an inch to one and a quarter inch in diameter, bright or dusky red in colour, gradually shading off into the surrounding skin, tender to the touch, and perhaps pitting slightly on pressure. These occur most often over the whole length of both tibiæ, and not infrequently over both ulnæ. Though rare in other parts, they may be seen on the calf, on the thighs, over the scapulæ, and over the condyles of the humerus. They come out more or less in crops, last seven or ten days, and gradually subside with bruise-like staining. They may become soft and fluctuate, but never suppurate. They are most common in children and people under twenty years of age; and more frequent in girls than in boys. The onset is preceded by some malaise, pains in the joints, and slight pyrexia. Though sometimes associated with rheumatic fever, it is very doubtful whether the rheumatic toxin is its only cause. But there are grounds for regarding it as infectious in character.

Treatment.—Sodium salicylate has been largely used for erythema nodosum, on the hypothesis of its rheumatic origin; but iron in combination with saline purgatives is often quite successful—*e.g.*, the sulphates of iron and magnesia, with peppermint water.

ERYTHEMA PERNIO

Pernio, or chilblain, is a superficial dermatitis, affecting the toes, sides of the feet, and the fingers, as a result of cold in people of defective circulation, and especially in children. There are patches of dusky redness, with itching, smarting, and pain, which come on frequently with the cold winter weather, and may only completely subside with the return of spring. In severe cases, or if irritated by friction or injury, they may vesicate or form indolent ulcers. They should be prevented, if possible, by warm clothing, sufficiently loose boots, and active exercise, such as running, skipping, dancing, and skating.

Treatment.—When they occur they should be rubbed with lin. camph. co., with or without lin. belladonnæ; or tr. iodi may be painted on the feet, or unguentum iodi rubbed in. Tr. iodi, decolorised with half its quantity of liq. ammoniæ, may be used for the hands. Calcium chloride in 15-grain doses may be given internally. Immersing the hands or feet in an electric bath is often of great use.

ERYTHEMA INTERTRIGO

(*Eczema Intertrigo*)

This is the inflammatory redness which occurs in the folds of the skin in fat people, especially under the mammæ, between the buttocks, and between the thighs and the scrotum or labia in children. The redness corresponds closely to the parts of skin that are in contact; the surface is raw, denuded of the upper layers of the epidermis, and it secretes a whitish turbid fluid, different from the yellow serum or sero-pus of eczema, and not drying into crusts unless mixed with the medicinal substances applied to it. In children it is no doubt aggravated by contact with napkins wetted by the urinary and fæcal discharges; and, as it not infrequently coexists with parasitic stomatitis or thrush, the mother generally regards it as thrush which has passed through the child. It may co-exist with ordinary eczema. It must be distinguished from syphilitic eruptions, which usually spread beyond the limits of the contact of skin with skin, or skin with napkin, and may be quite dry.

Treatment.—The parts should be separated, and contact with secretions and wet napkins should be prevented. This is best done in children by spreading zinc or boric ointment on narrow strips of lint and carefully laying them over the thighs and scrotum or labia, so that they are protected from one another and from the napkin. The lint should, of course, be changed directly it is wetted or soiled by the motions. In slight cases fuller's earth or zinc oxide may be dusted over the surface. Any defect of health should be treated, especially diarrhœa, the acrid discharges of which may intensify the trouble. In adults, powder of zinc oxide, mixed with two or three parts of starch, or of boric acid and kaolin, may be dusted over the part and a piece of lint placed in the fold; or ointment of zinc or boric acid may be used.

OTHER FORMS OF ERYTHEMA

Erythema simplex is redness in patches, with little or no infiltration, and not very persistent. *E. fugax* is a still more fleeting lesion. *E. læve* is the hyperæmia which occurs in the tense skin of anasarcous limbs. It may go on to deeper dermatitis and sloughing.

Erythematous eruptions also form part of the epidemic diseases known as *pellagra* and *acrodynia*. The former has already been described (*see* p. 112); the latter was epidemic in Paris in 1828–29, and has since been seen but rarely. Erythematous patches starting in the hands and feet were followed by pigmentation and desquamation; and were associated with hyperæsthesia, anæsthesia, cramps, and paresis.

LUPUS ERYTHEMATOSUS

This disease occurs mostly in adults, especially between the ages of twenty and forty, and is more common in women than in men. It often begins as a red patch on the centre of the nose, and spreads thence right and left over each side of the nose to the cheek, on which it forms a broad patch. It has then much the outline of a butterfly or bat—a very characteristic feature of the disease. The patch is red, injected, desquamating, with obviously enlarged and distended sebaceous glands; the edge is rather sharply defined, irregular, and slightly raised. As it spreads at the periphery the centre becomes pale, and ultimately scars, without preceding ulceration. The ears and the scalp are sometimes attacked, and in the latter case the hair falls out, and permanent baldness may result. The backs of the hands, and less often the arms, legs, and trunk, are also seats of the disease.

Two varieties are described, one in which single spots enlarge at the periphery (*discoid form*); the other, in which crops of spots appear, and coalesce to form large areas (*disseminate form*).

Lupus erythematosus makes but slow progress, and lasts for years; it is free from pain, and itching is slight or none. Some cases have been seen of an acute kind, in which a large part of the body was rapidly covered, and death ensued; but, as a rule, the disease has little or no effect upon the health. The condition is one of inflammation closely allied to the erythemata, but very persistent, and terminating in scar-formation. There is thinning of the epidermis, dilatation of capillaries in the superficial layers of the cutis, dilatation of lymph-spaces, infiltration with leucocytes and connective-tissue cells, and œdema of the skin-tissues. Ultimately atrophy takes place, and a thin scar results. Hitherto no constant connection with tubercle has been proved.

Treatment.—The local treatment should be by mild caustics and stimulants: Tincture of iodine painted on, ointments of pyrogallic acid (1 in 8), of ichthyol, of iodoform (1 in 16), or white precipitate, of yellow oxide of mercury, of zinc, lead, and mercury (*see* p. 1040), of litharge (Hebra's diachylon ointment), or of liquor carbonis detergens (1 in 16, Hutchinson). The more hyperæmic forms require milder preparations, such as calamine or zinc lotion; in any case the applications must be made regularly and perseveringly over long periods. The treatment by light rays has been less successful than in Lupus vulgaris. Internally, quinine, salicin, arsenic, and iron may render some help.

ROSACEA

(*Gutta Rosea*)

This is a condition of chronic dilatation of vessels, a persistent erythema, or the result of recurring erythema. It occurs in men more frequently than in women, and seldom before middle life. The most common cause is the frequent use of alcohol, or the kind of indigestion which is the result of excessive drinking and overfeeding; in women, ovarian and uterine disturbances are frequently the cause. It begins as an erythematous redness affecting the nose; repeated attacks lead to infiltration and permanent thickening of the skin. It spreads to the cheeks, the centre of the forehead, and the chin. The minute vessels become dilated, and are visible on the surface; the sebaceous follicles are distended with secretion, and inflame, so that eventually the affected part shows a deep red swollen and thickened skin, with dilated venules, and papules, pustules, and nodules of various sizes. The nose is especially the subject of great hypertrophy of the tissues, and may form a large lobulated tumour of an inch or more in diameter (*rhinophyma*). Congestion and inflammation of the deeper layers of the corium, accumulation of sebum, with inflammation of the sebaceous glands, and abundant growth of connective tissue, are the leading histological changes.

Diagnosis.—It is distinguished from *Lupus erythematosus* by the absence of scabs and the presence of pustules; and from *syphilitic* eruptions by the symmetry, and the absence of other characteristic lesions.

Treatment.—The errors of digestion must be corrected. Alcohol should be forbidden, the diet carefully revised, bismuth, alkalies, or bitter tonics administered, and a regular action of the bowels ensured. An ointment containing resorcin 20 grains, and zinc oxide 20 grains, to the ounce, or ichthyol ointment (20 or 30 grains to the ounce), may be used; or calamine and oxide of zinc lotion. Ichthyol internally (5 to 10 grains) is also of value. The local treatment may be similar to that of acne vulgaris, but the stimulants must be milder. Dilated vessels may be incised, and touched with silver nitrate, but large excrescences will require removal with the knife.

URTICARIA

Urticaria (*urtica*, a nettle) has a close alliance with erythema. The eruption often comes out suddenly, and consists of firm, round, convex, or lenticular elevations of the skin from a quarter of an inch to an inch in diameter, at first pink, and soon becoming white in the centre. These are called pomphi or wheals. They are scattered or closely crowded over the part affected, and are not symmetrical. They may arise very rapidly, and subside in a few

hours or a day (*U. acuta*), or they last longer, or recur frequently (*U. chronica*). Sometimes the elevations are quite small (*U. papulata*); in rare cases as large as a walnut or hen's egg (*U. gigas*). Rarely a small vesicle may form on the surface of the wheal (*U. bullosa*). Urticaria is accompanied with intense itching, so that the patient cannot forbear from scratching himself, and thus, no doubt, the lesion is considerably aggravated.

Causes.—Urticaria is due in a large number of cases to the action of poisonous substances, either applied locally, or circulating in the blood after absorption from the alimentary canal. Among the former we have the poison of the stinging nettle, the sting of bees and wasps, and contact with jelly-fish and certain caterpillars. U. papulata, which occurs especially in children, is perhaps often due to the irritation of fleas and bugs.

Among the latter are (α) certain foods, especially shell-fish, the less digestible meats, pork and sausages, mushrooms, and some fruits; (β) certain drugs, namely, copaiba, cubebs, quinine, and others. In (γ) some general disorders, such as gout, indigestion not specially related to the above ingesta, menstruation, lactation, pregnancy in women, and asthma there is also the possibility of toxic substances being in circulation. But there are chronic cases in which no such cause can be traced.

In some individuals direct mechanical irritation will cause a local infiltration to take place almost at once, so that the scratch of a pen or the nail upon the skin is sufficient to raise a linear ridge; and thus figures or letters can be traced upon the skin lasting for some minutes (*factitious urticaria*).

Wright states that in many cases of urticaria there is a defective coagulability of the blood, and a deficiency in the lime-salts.

Angeio-neurotic œdema (*see* p. 663) is perhaps allied to urticaria.

Treatment.—The cause must be looked for and removed. In acute cases clearly due to ingesta an emetic is indicated. In other more chronic cases, the diet should be carefully considered, and search should be made for any particular article of diet which may be responsible; the bowels should be regulated, and defects in digestion should be met by suitable treatment. In gouty people, colchicum and salines should be given; and in others, general tonics may be desirable. Chronic urticaria is sometimes very intractable; quinine, with saline laxatives, arsenic, or ichthyol (3 gr. to 5 gr. in pill or capsule) may then be of value. Calcium chloride or lactate may also be tried, and with a similar object milk, which is rich in calcium salts, may be given freely. The severe itching requires local treatment. Scratching must be prevented, and one of the following applications should be used: Alkaline baths (sod. bicarb. 2 to 6 oz. in the bath), or alkaline lotions (sod. bicarb. ℥j or ℥ij to ℥vj), calamine and zinc oxide lotions, hydrocyanic acid lotion (℥iij of dilute acid in aq. ℥xx), and lead lotion. Antiseptic lotions are also valuable: Liq. carb. detergens (℥ij or ℥iij to aq. ℥viij); terebene (℥j to ℥viij); sanitas and water, equal parts; a saturated

solution of benzoic acid; carbolic acid (ʒss or ʒj to ʒviij), and others.

URTICARIA PIGMENTOSA

This is a somewhat rare form of disease, and its connection with the above is at least doubtful. It begins in early infancy, and consists of round or oval maculæ and raised patches of dark brown, brownish-red, yellow-brown, lemon-yellow, or fawn colour. The patches may remain unchanged for years, but some after a time disappear, leaving only pigmentation. The majority of cases have been accompanied by itching, with the result of producing secondary wheals, factitious urticaria, erythema, and enlargement of the lymph-glands. The chief histological features are the œdema of the cutis, increased pigmentation of the deeper layers of the epidermis, and a great abundance of *mast-cells* in the cutis and subcutaneous tissue, especially around the vessels, hair-follicles, and sweat-glands and their ducts.

Treatment has hitherto had little effect, beyond the relief of itching. Many cases have lasted twenty or thirty years; in a few the eruption has subsided, leaving stains or faint scars.

PEMPHIGUS

This is an eruption of bullæ or blebs occurring as a primary lesion. Bullæ have already been described as occurring in some forms of erythema, and they may be caused by burns, irritants such as blisters, and by the itch-acarus; but in pemphigus, which is a rare disease, the bulla is the primary and chief lesion.

PEMPHIGUS VULGARIS

Ætiology.—Pemphigus vulgaris occurs at all ages, but is more common in children than adults, and in females than males. Heredity has been noticed a few times, but beyond this very little is known of its causation.

Pathology.—There is inflammation of the papillary layer of the skin, with fluid effusion lifting the epidermis; but whether the bleb forms in the layers of the rete or below seems yet uncertain. Diplococci and staphylococci have sometimes been found in the lesions of pemphigus. Crocker pointed to the occurrence of bullæ in diseases of the central nervous system as suggesting a possible pathology for pemphigus. Eosinophilia is often pronounced.

Symptoms.—The disease usually runs a somewhat chronic course. The eruption may be preceded in some people by chilliness, nausea, or pyrexia; then the bullæ appear at one or other part of the body, small at first, gradually increasing in size, tense, hemispherical, with clear yellow or slightly turbid contents. The fluid

is an albuminous serum, and the turbidity is due to the presence of leucocytes. Around the bleb the skin is at first quite normal, but a narrow pink areola is acquired later, and increases in proportion to the opacity of the fluid. After a few days the fluid is absorbed, or the bleb ruptures, and shrinks down on to its base. From this it is subsequently shed; it leaves a mark which is injected and afterwards slightly stained but rarely or never scarred. Sometimes the bleb contains pus or blood, and after its rupture the base may be covered with yellow lymph, or may slough.

The number of bullæ in any case is very variable. There may be but few in one part, or isolated bullæ in different parts of the body; or the whole surface may be thickly covered by blebs, which come out in successive crops, lasting only a few days each, but keeping up the disease for weeks and months. Nearly every part of the body may be affected, but the hairy scalp least of all. Rarely the conjunctiva is attacked with pemphigus, which is followed by contraction (*essential shrinking*), producing deformity and ultimately blindness.

The amount of general disturbance is greatest in children and old people, and is proportionate to the extent of surface involved. Young adults with few bullæ are not materially affected. Sometimes there is severe itching, with all the secondary results which follow scratching, such as wheals, eczema, and pustules, while the contents of the bullæ are likely to become more purulent (*P. pruriginosus*). Many cases are very intractable, and some end fatally.

Acute pemphigus is much more rare than the chronic form; the course is rapid and sometimes fatal, and pyrexia is present. Some such cases have occurred in butchers after wounds on the fingers, and diplococci have been found in the fluid of the bullæ (Pernet and Bulloch).

Bullous eruptions readily occur in infants, and have often been called "pemphigus neonatorum." But many cases of bullous formation in infants are due to syphilis; and others occur apparently as the result of infection from without: thus it has been seen in association with impetigo contagiosa in the mother, nurse, or midwife, and the same organisms have been found in both lesions. In infants also scabies may lead to bullæ, as well as vesicles.

Treatment.—Arsenic is of great value in chronic cases of pemphigus vulgaris, and should be given perseveringly in full doses. It is not generally so useful in acute cases. Locally some relief from discomfort may be obtained by the use of calamine lotion, zinc ointment, zinc oxide dusted on, or similar soothing applications. Tonics, quinine, iron, &c., and sufficient food are desirable also.

PEMPHIGUS FOLIACEUS

This is a very rare and fatal form of pemphigus, in which the whole surface of the body is gradually involved. The blebs which form are flaccid and flat, never tense and hemispherical. Their contents are turbid, and when these escape an inflamed excoriated

surface is left; to this the remains of the bullæ adhere, forming thin flakes, the under surface of which is moist with an offensive secretion. If the flakes are removed there remains a red, raw, secreting surface, not unlike eczema rubrum. When the whole surface is affected it is mostly covered with the adherent epidermis, and with raw patches at intervals; then also the occurrence of blebs is not easy to observe, as they form under the existing epidermis and soon rupture. The course is slow, with remissions and relapses, it may be with healing of the skin in parts; but eventually the disease is fatal by exhaustion or intercurrent disease.

Treatment.—Drugs have no influence; immersion in a warm bath gives temporary relief.

PEMPHIGUS VEGETANS

is another rare variety, in which the mouth is first affected, then bullæ of ordinary type form on the skin, ulcerate, and remain unhealed for a long time. The characteristic feature is that in moist situations, like the axillæ, groins, and gluteal folds, fungating papillary growths form on the site of the ruptured blebs, project a quarter to half an inch above the surface, and secrete an offensive muco-purulent fluid. Severe prostration ensues, and the cases end fatally. Some relief may be obtained by local antiseptic applications.

EPIDERMOLYSIS BULLOSA

This is a condition perhaps allied to pemphigus, in which the epidermis separates very readily from the cutis, on slight injury, such as rubbing vigorously, or pinching; and flaccid bullæ slowly arise, of which the contents may be blood-stained. Thin shining scars may be the result of the lesions: and often the nails are shed. The disease arises in early life, may be hereditary, and is but little amenable to treatment.

HERPES

This name has been given to certain vesicular diseases, but it is not easy to give a definition that will cover all One may say that the vesicles of herpes are generally smaller than those of pemphigus, are seated upon an inflamed base, and terminate by scabbing. The diseases for which the name is still retained are herpes zoster or zona, herpes labialis, and herpes preputialis.

HERPES ZOSTER

(Zona. Shingles)

This is an eruption of vesicles, arranged in groups, which always correspond in position to the distribution of a cutaneous nerve.

The name zona, or girdle, is taken from the most common or intercostal variety, in which the groups of vesicles extend from the spine round one half of the body to the middle line in front. The eruption is preceded sometimes by pain, tingling, or smarting, and it may be a little malaise or slight pyrexia; then appear groups of closely set papules, forming red patches, one or two inches in diameter; and upon these the vesicles quickly arise, with thin walls, clear contents, not very tense, and, when numerous, acquiring a polygonal form from mutual compression. The patches do not all appear simultaneously —for instance, one may form first near the spine, then later one in the axilla, and later again one near the sternum; some patches also —that is, the later ones—may fail to produce any vesicles, the process, as it were, subsiding early or aborting. After a time the contents of the vesicles become opaque or milky, and the vesicle dries into a scab which drops off, leaving a red stain. The milkiness may amount to the formation of pus, and the superficial layer of the skin may be destroyed, so that scars result. Scars may form in each group, but not in every vesicle of a group. Quite rarely extensive sloughing of the skin takes place, leaving deep ulcers, which heal slowly. The patches are distributed in the course of a nerve; on the trunk they form a band from two to four inches broad, generally somewhat more horizontal than the true course of the ribs, and the vesicles may transgress the middle line both in front and behind.

H. frontalis occupies the area of the supraorbital nerve on the forehead and scalp, and there is often conjunctivitis. *H. cervicalis* lies over the neck, clavicle, and deltoid; *H. brachialis* follows the course of the nerves of the arm; and other similar groupings on the abdomen, thigh, and leg are occasionally seen. An intercostal zona may be accompanied by herpes of the inner side of the arm (intercosto-humeral nerve), or a gluteal by an anterior crural, representing posterior and anterior branches of the lumbar nerves. The eruption is nearly always unilateral, and its bilateral occurrence has very rarely been recorded.

The duration of the eruption is from four to ten days, but the disease does not always end here. Especially in old people, neuralgic pain in the course of the affected nerve may continue for months or years, and be a source of serious trouble; and in a few cases paralysis of associated motor nerves or nerve-fibres has been seen, most often of the seventh or third nerve in facial zoster, but also of the nerves to the deltoid and abdominal muscles.

Pathology.—The eruption itself is an inflammation of the papillæ and corium, followed by effusions into the layers of the stratum mucosum; but the close relation to cutaneous nerves has naturally led to investigations as to their condition, and in a great number of instances lesions have been found. The most frequent is inflammation of the ganglion on the posterior root of the spinal nerve, and of the nerve below it; but inflammation of the sensory root above the ganglion, peripheral neuritis, neuromata, and hæmorrhage into the Gasserian ganglion have also been recorded. While sometimes due

to local causes (disease of the ribs), it more often presents all the characters of an acute infectious disease, and attention has been called to the simultaneous occurrence of vesicles in parts remote from the nerve concerned (*aberrant* vesicles) in support of this. Herpes zoster also occurred in some of the cases of arsenical poisoning in beer-drinkers in 1900.

Diagnosis.—This depends on the unilateral group of vesicles, corresponding to the distribution of a nerve.

The **Prognosis** is favourable, but the probability of scarring, and the tendency to troublesome neuralgia in elderly patients, must be remembered.

Treatment.—Nothing will check the disease; but we should try to protect the vesicles from injury and from rubbing by the clothes, and to allay any irritation, tingling, &c. This may be done by applying powdered zinc oxide with starch powder, to which a little pulvis opii may be added if the pain is severe, and covering it with cotton-wool. When the vesicles burst, or the skin ulcerates, zinc ointment or boric ointment may be applied. For the severe pains afterwards, arsenic, antipyrin (in 10-grain doses), phenacetin (10 gr.), quinine, or sodium salicylate should be given. But morphia, either internally or subcutaneously, is often required. Menthol may be rubbed in; and blisters over the origin of the nerve and the continuous galvanic current also give good results.

HERPES FACIALIS, OR LABIALIS

This occurs as groups of vesicles forming rapidly upon an inflamed base. The contents are clear at first, then turbid, and afterwards dry into a scab, which falls off, leaving scarcely a mark. It affects the lips, the alæ of the nose, and the adjacent cheeks, is usually bilateral, and lasts from five to ten days. It mostly occurs in association with some acute febrile disease, especially with croupous pneumonia, of which it is sometimes considered diagnostic; but this is not so, as it happens in ordinary catarrh and bronchitis. It occurs in diphtheria and in relapsing fever, and one sometimes sees it without any other recognisable disease, other than a sharp pyrexial attack, with high temperature and rigor. It not infrequently recurs in the same person. Local sedative applications, such as zinc lotion or calamine lotion, are all that are required.

HERPES PREPUTIALIS

This closely resembles the preceding. It consists of a vesicle or a group of vesicles on an erythematous base, occurring on the inner side of the prepuce, less often on the outside, on the glans, in the meatus or even in the urethra. But it is seen sometimes on the labia, nymphæ, and pubes in women. It is often preceded by some local disease, such as gonorrhœa, soft chancre, or stricture of the urethra, and it is of importance, because the vesicles rupture early, and form

small ulcers, which may be mistaken for chancres. Like herpes facialis, it is apt to recur. The treatment is to keep the parts thoroughly clean, and to apply lead lotion on strips of lint, or dust starch and zinc oxide over the vesicles, and separate the parts with lint. Iodoform or lotio nigra on lint may be used where a sore has formed.

DERMATITIS HERPETIFORMIS

Dühring, who introduced this term, gives the following definition of the disorder to which he applies it: "An inflammatory, superficially seated, multiform herpetiform eruption, characterised mainly by erythematous, vesicular, pustular, and bullous lesions, occurring usually in varied combinations, accompanied by burning and itching, pursuing usually a chronic course with a tendency to relapse and recur." This includes cases hitherto called *hydroa*, probably some of the cases recorded as pemphigus, erythema iris, and pemphigus iris. It covers *herpes gestationis* or *pemphigus gestationis*, a bullous eruption which occurs during pregnancy, affecting more or less the whole of the body, disappearing with delivery, and recurring generally in future pregnancies. D. herpetiformis thus presents the most varied lesions over the body at the same time, in one place patches of erythema, in another urticarious wheals, and in a third bullæ like those of pemphigus; and there may be pyrexia with it. The disease is very little amenable to treatment; the lesions may spontaneously heal at one part, and break out in another. The itching is exceedingly troublesome; but the patient's health and nutrition are generally maintained, and many cases ultimately recover.

The lesion is an acute inflammation of the papillary layer of the corium with the formation of vesicles directly beneath the epidermis; and the extravasation of enormous numbers of polymorphonuclear and eosinophilous leucocytes. The proportion of eosinophile corpuscles in the blood is also largely increased, up to 10 or 15, or even 30 per cent. of the leucocytes.

Treatment.—Arsenic, quinine, salicin, and nux vomica are of value internally, and locally sulphur ointment and ichthyol preparations (*see* p. 1041) may be employed.

CHEIROPOMPHOLYX

This name, meaning bullæ on the hand (from χείρ, a hand, and πομφόλυξ, a bubble), was given by Hutchinson to an affection which is not very common, at least in its most marked stages. The subjects are young people, more often women who are slightly out of health, and who are inclined to perspire about the hands; it occurs also mostly in the heat of summer. After a little tingling and itching there appear small translucent spots on the skin of the sides, or tips or bases of the fingers, and in the palm of the hand.

These spots look like, and indeed are, vesicles beneath the horny layer of the skin, which, however, is not raised by them until they reach a very great size. They may be isolated, but when abundant form groups, half an inch to an inch or two in diameter, of closely packed vesicles, so that the affected skin, still flat, looks as if it were formed of boiled sago grains. If one of these is punctured a clear or very slightly turbid, perhaps ropy, fluid exudes, which is neutral or alkaline, and contains albumin. There is no reddening of the skin unless just at the margin of a large patch. In later stages the septa between the vesicles may disappear. Several vesicles run into one another, and the horny epidermis is raised into large blebs or bullæ projecting beyond the skin. After a week or more the contents are absorbed, and the skin forms a dead flake, which is shed; it leaves a pink new skin, which gradually assumes the normal appearance. The only other parts of the body subject to it besides the hands are the feet, and they are not always, and never so badly, affected.

Anatomically it is seen that the vesicles form in the stratum mucosum from exudation of serum among the cells forming it. It was once described as *dysidrosis*, from the belief that the vesicles were obstructed sweat-ducts, but the alkaline, albuminous nature of the fluid disproves this.

The patient, as a rule, readily recovers, but the disease is liable to recurrence. Its recognition is not difficult; the curiously aggregated vesicles within the skin are characteristic.

Treatment.—A tonic treatment should be adopted with the local application of sedative lotions and ointments, such as those of lead and zinc. Too irritant preparations may inflame the skin, and convert the disease into a true eczema.

TOXIC DERMATITIS

Many forms of dermatitis can be traced to the direct application of poisonous materials or to their internal administration. Such, for instance, in a mild form is the familiar urticaria of the stinging-nettle (*Urtica dioica*), and in a much more severe form the erythematous rash produced by contact with the leaves of the Primula obconica, and with the juice of plants of the order *Anacardiaceæ*, to which Rhus toxicodendron and the Indian marking-nut belong. I described a case of the erythematous and bullous results of the latter many years ago (*Med. Times and Gaz.*, Nov. 6, 1875). The results of the presence of toxins and allied poisons in the blood are seen in the erythematous eruptions of the exanthems, in the occasional eruptions of pyæmia and septicæmia, in eruptions after vaccination and in the erythematous rashes which sometimes follow the injection of diphtherial and other antitoxins. *Uræmic dermatitis* is a toxic form which is occasionally seen in chronic Bright's disease towards the end of the illness (*see* p. 912). It

often begins as papules or larger elevated patches of red inflamed skin, which ultimately coalesce, so that the whole body may be covered with red thickened skin. Subsequently desquamation takes place, and some cases have a close resemblance to exfoliative dermatitis. Lastly, we have the poisonous effects of certain drugs when given in undue quantity or when unduly retained by inadequate elimination by the kidneys.

ERUPTIONS CAUSED BY DRUGS INGESTED

The eruptions produced by drugs are erythematous, urticarial, vesicular, bullous, purpuric, or in some other form, but the first four varieties are more common, and especially the first—namely, erythema. The following are the most important:

Antipyrin.—A red, papular or morbilliform eruption over the greater part of the body, sometimes with itching and subsequent desquamation. Purpura has also been seen.

Arsenic.—Urticaria, erysipelatoid rash, or small papules. Herpes zoster has occurred during the use of arsenic. The long-continued use of arsenic has caused a general pigmentation of the skin, and in psoriasis the healed patches sometimes become very deeply stained. *Keratosis*, or thickening of the horny layer of the epidermis, especially affecting the soles of the feet and palms of the hands, also occurs, and was seen in the arsenic-poisoning of beer-drinkers in 1900.

Borax and Boric Acid.—Inflammations of the skin have occasionally followed the internal use of borax, as well as its application to the surface, and to internal cavities. The eruptions are erythematous, and sometimes bullous, or even hæmorrhagic.

Bromides.—An acneiform eruption is common as the result of the use of bromides in epilepsy—the pustules are commonly discrete, and occur on the face, chest, back, or scalp, and around the hair follicles on the thigh. More extensive lesions occur in exceptional cases in children on the face and limbs; these are large, oval or circular, much raised patches of deep red colour, covered with a number of pustular points, or the thick scab which follows their rupture. The substance of the patch is soft; it mostly subsides, and the scab is detached, without leaving any scar, but only a rather persistent stain. The lesions often begin some days after the bromide has been stopped, and their appearance is favoured by disease of the kidneys hindering elimination of the drug. Arsenic internally promotes their cure, and if given with the bromide may prevent their occurrence. Erythematous, papular, and bullous eruptions also occur.

Chloral.—Erythematous eruptions, diffuse redness or red papules, and occasionally purpura. They occur mostly after long-continued use of the drug.

Copaiba.—Erythema, consisting of bright-red, roundish or irregular patches, slightly raised above the surface, here and there

confluent, somewhat like measles, covering the arms, legs, trunk, and face. Purpura, vesicles, and urticaria are occasionally present. Desquamation may occur after a persistent eruption.

Cubebs seems occasionally to produce a similar rash.

Iodides.—The eruptions are erythematous, pustular, vesicular, bullous, or purpuric (*see* p. 848). The erythema is papular, and occurs over the trunk, face, and limbs. Pustules are seen like those of the bromide rash, but smaller in size when discrete; and the confluent forms are less common, and tend to be more bullous. Sometimes large bullæ occur, with a very narrow areola around them, and clear serous contents. Like the bromide eruption, it may be delayed for some days after the drug has been stopped, and is more likely to appear if the kidneys are diseased. The addition of arsenic, or aromatic spirits of ammonia to an iodide mixture, or taking the dose in half a tumblerful of water, may be tried to prevent its occurrence.

Quinine.—Erythematous rashes are most common, either diffuse or papular; an urticarial form is next most frequent; both of these produce severe itching, and erythema may be followed by extensive desquamation. Purpuric, vesicular, and bullous rashes are less often seen.

Other drugs that have more or less frequently caused rashes, mostly of an erythematous or urticarial type, are belladonna, cannabis indica, potassium chlorate, chloroform (inhalation), cod-liver oil, digitalis, iodoform, mercury, morphia, opium, phenacetin, phosphoric acid, salicylic acid, santonin, strychnia, stramonium, sulphonal, tar, terebene, and turpentine.

The **Treatment** should be the withdrawal of the drug and the use of astringent lotions, such as those of subacetate of lead, oxide of zinc, or calamine.

ERUPTIONS CAUSED BY DRUGS APPLIED EXTERNALLY

Many substances are applied externally in order to cause erythema and vesication for their therapeutic effects: such as cantharides, capsicum, mustard, croton oil, and turpentine. Belladonna, iodine, sulphur, mercury ointments, arnica, chrysarobin, and pyrogallic acid are also likely to cause irritation if too long applied on sensitive skins.

TRAUMATIC AND SOLAR DERMATITIS

Apart from surgical conditions, *traumatic dermatitis* may be recognised in the marks of scratching, such as are seen in pruritus, jaundice, scabies, and phtheiriasis, to which the reader is referred. These effects are in part due to repeated infections by pus-organisms.

Another form of traumatic dermatitis is that intentionally produced in order to *feign* disease: an event most common among young women, who may use nitric acid, mustard, cantharides, iodine, or other irritant. The site of the lesion is generally the breast or a limb, at least a part accessible to the right hand: the lesion is generally redness with or without vesication, or pustulation.

Solar dermatitis (formerly called *eczema solare*) is well known to follow unwonted exposure to the rays of the sun as reflected from the cricket field, the river or sea, and especially from Alpine snow-fields. There is intense redness and swelling, with formation of vesicles and bullæ; accompanied by itching and smarting pain, and followed by free desquamation and pigmentation. Bowles asserts that this is due rather to the light rays (*actinic*) than the heat rays, and his contention appears to be borne out by the effects of light used in the treatment of lupus, &c. Of analogous origin is the severe and persistent dermatitis which results from prolonged exposure to the *Röntgen rays*.

ECZEMA

Eczema (ἐκζέω, to boil over) is a superficial inflammation of the skin, produced without obvious external cause, and presenting a great variety of lesions, the most common of which is vesication, followed by destruction of the superficial layers of the epidermis, and the prolonged secretion of serum. In this, the typical form of eczema, the eruption begins with some itching or smarting at one spot, which then becomes red, and several minute vesicles form upon it, containing clear yellow serum. They soon rupture, and discharge the serum; and the abraded spots thus produced extend and coalesce by the temporary formation, not always very perfect, of fresh vesicles. The secreted fluid is albuminous, and stiffens linen; it is mostly clear yellow, but may be more or less opaque from corpuscular elements. After flowing for a little, it dries up into translucent yellow or opaque whitish or greenish-yellow crusts, which adhere to the surface until detached by accident or lifted by discharge underneath. When they are removed fresh secretion takes place, again drying up into crusts, and this process may go on indefinitely until spontaneous cure or treatment ends it. Sometimes an adherent crust will grow to a great thickness from the secretion underneath. Eczematous patches enlarge by extension at their periphery, which generally shades off

into the adjacent skin; and they are often surrounded by other patches, each from a quarter to half an inch in diameter, with a small pin-point vesicle, abrasion, or crust in the centre.

Healing takes place by a gradual cessation of the secretion, and covering of the abrasion with sufficient epidermis. This may take place spontaneously under a crust, which may remain long after recovery is advanced. The skin is, however, not normal for some time. Redness and thickening persist, and the epidermis forms large flakes and scales, which are from time to time detached.

The patches of eczema are variable in size, from mere spots to large continuous areas. It occurs in nearly every part of the body; but with especial frequency on the face, ear, scalp, neck, flexure of the elbow, front or back of the forearm, wrist, groin, inner side of the thigh, and flexure of the knee.

Locally, eczema gives rise to severe itching, smarting, or burning, and in certain positions to pain on movement. The general condition of the patient in eczema may be but little affected. In acute cases, with extensive patches, there is some febrile reaction; in many instances the eruption is coincident with, and no doubt induced by, a general malaise, or anæmia, or temporary depression of health; and in prolonged chronic cases the health may be slightly affected as a result of the eczema.

Variations in its course give rise to special names. *Acute* and *chronic* eczema are distinguished by their intensity and duration; but long-continued cases may have frequent acute outbreaks. If the inflammation is intense, with much redness and profuse secretion, it is called *E. rubrum*, or *E. madidans*. In a later stage, or with a less active eruption, the secretion is diminished or absent, and the dermatitis results only in the formation of layers of epidermis, which are successively shed (*E. squamosum*).

Seborrhoic Eczema.—Unna pointed out the frequent association of eczema with *seborrhœa*, and although his inclusion of all forms of the latter under the head of eczema is not generally accepted, it is clear that seborrhœa is often the precursor of a typical eczematous lesion. The lesions may consist of red or infiltrated patches, with greasy yellow scabs, or of completely eczematous lesions, with raw surface, secretion, and scabs. The scales and crusts are yellow in colour, and fat is present in them. Under the head of *E. seborrhoicum* have been included also extensive areas of redness with slight greasy scales on the trunk; moist eczematous lesions under the mammæ in women, and in the axillæ; some circinate and squamous lesions in the palms of the hands, and some forms of dermatitis about the groins, pubes, and scrotum.

E. papulatum is a papular form of eczema, which was formerly described as *lichen*. It is common on the backs of the forearms and on the back.

E. rimosum is the name given to a chronic form in which there is much infiltration and thickening of the skin, with a thin scaly

epidermis, only a few scattered discharging points, but several fissures running deeply through the epidermis into the corium, not infrequently bleeding, and excessively sore and tender. This often occurs about the wrists and the lower parts of the forearms.

In other cases the thickening and hypertrophy of the skin form the chief trouble. It is quite rigid, and cannot be pinched up. The surface is marked out by small furrows into diamond-shaped areas, and it is white, or powdery from half-detached epidermis. A warty condition may also occur from hypertrophy of the papillæ (*E. verrucosum*).

Ætiology.—Eczema occurs equally in both sexes, and at all ages of life. A number of external irritants, thermic, chemical, or mechanical, give rise to a dermatitis which has many of the features of eczema. Among these are exposure to the sun (*see* p. 1036); and the application of various drugs to the skin—*e.g.*, mercurial ointment, and some very alkaline soaps—produces an allied condition. In certain trades the hands are constantly irritated, chemically or mechanically, by the substances handled. "Grocer's itch" is an eczematoid dermatitis of the hands and wrists from contact with sugar and other groceries. Discharges from the ears, nose, or other parts, and friction of the clothes, may also be causes of the same. In many of these instances the lesion is no more than a traumatic dermatitis; and in some of them the inflammation may extend deeper into the subcutaneous tissue, and produce larger vesicles or blebs than is common in eczema. They are more deserving of this name when this disease continues after the removal of the cause, or extends beyond the area immediately affected by it. Either suggests the existence of some special tendency on the part of the patient, or some external agency, as, for instance, a micro-organism.

That seborrhœa is a frequent antecedent of eczema has been already shown.

A tendency to eczema, though rarely *hereditary*, certainly seems to exist in some persons. From time to time eczema breaks out on the skin, it is cured by appropriate treatment, and again recurs on slight irritation, or on some slight alteration of general health, or it may be, to all appearance, quite spontaneously. Amongst the conditions of ill-health to which eczema is in some cases attributable, are various kinds of dyspepsia, intestinal disturbances, whether constipation or diarrhœa, especially in young children, anæmia, the condition of feeble vitality of some children, mental strain and nervous shock (M. Morris), and gout.

Eczema is not itself due to micro-organisms; but the abraded surface of the skin is very liable to infection by pyogenic organisms, especially streptococci and staphylococci; the secretion then becomes purulent, the scabs are opaque and greenish-yellow in colour, and the lesion is said to have become *impetiginous*. The change renders it more resistant to treatment.

Anatomy.—In the epidermis the cornification is irregular, the horny layer is thinned, and the cells of the stratum mucosum approach the surface; there is also proliferation of the prickle-cells, which are surrounded and infiltrated with fluid. In the papillary layer of the corium the vessels are dilated, and there is an increase of fluid and leucocytes in the tissue. In chronic cases the stratum mucosum dips down deeply between the papillæ, which are elongated to a proportionate extent.

Diagnosis.—The red, raw surface exuding serum or sero-pus which dries into scabs, is characteristic of eczema, and is of more use in diagnosis than the vesicles, which are often of temporary duration. *Scabies* (itch) may be mistaken for eczema; the lesions are mostly scattered pustules or vesicles (even bullæ in some instances), and not continuous patches. It occurs in certain situations, the wrists and fingers in adults, the toes, feet, and genitals in children. The "runs" or burrows, if seen, are conclusive evidence of the itch-acarus. Scabies may set up a secondary eczema. Eczema resembles *sycosis* when it is confined to the hairy parts of the face; the lesions are more superficial than those of sycosis, and not limited to the hair-follicles in the same way; the weeping on removal of crusts comes obviously from intervening skin. *Psoriasis* may be imitated by a dry scaly eczema; patches of psoriasis are more sharply defined, more uniform in shape, round or ringed, covered with thicker, drier, whiter scales; and the distribution is characteristic in many cases. *Tinea circinata* produces circular patches or rings on the face or elsewhere, which look like a dry eczema; their small number and their circular form should make one suspicious, and the association of ringworm of the scalp, or a microscopic examination, will complete the diagnosis.

Prognosis.—In its acute and subacute forms eczema is amenable to treatment, but many chronic varieties are intractable and last for years. There is a strong tendency to recurrence, which may show itself after long intervals. Only in young children and very old people is it likely that an extensive eczema will help to a fatal termination.

Treatment.—Local treatment is of the first importance in this disease. It is desirable, in the first place, that the eruption should be protected from every sort of irritation. Scratching, alternations of temperature, cold air, washing with plain water, or with alkaline soap and water, will all increase vascularity and secretion, and delay healing. The patient should abstain from scratching, and young children should have the hands tied in gloves, or fixed to the side of the cot. The part should only be washed with thin gruel and water, or with oatmeal and warm water. If there are crusts upon the skin they should be removed so that substances can be applied directly; for this purpose, boric acid lint steeped in hot water or strips of lint soaked in olive oil may be left in contact for three or four hours, so as to soften the crusts, which may then be carefully removed.

Various local applications serve both to protect from the external air and to modify the condition of the part. Simple oily applications are sufficient, by protection, to promote the healing of some mild cases. But in nearly all cases benefit is derived from the use of sedative and astringent or antiseptic applications; the last are especially necessary when the lesion has become impetiginous. Ointments, lotions, and powders are used. On the whole, ointments are more generally beneficial, since they can be kept more constantly applied, and lotions must be covered with some impervious material (gutta-percha or oiled silk), when the part becomes unduly heated and sodden; otherwise they evaporate, the lint or rag adheres to the part, and considerable irritation is the result. But if there is much secretion on large surfaces, and if the dressings can be constantly looked to, lotions are best. If ointments are used, they should be not merely smeared over the part, but spread upon lint and firmly and uniformly applied, so as to get complete contact; they should, as a rule, be changed twice a day, and the excess of old ointment should be gently removed with a soft cloth, or by washing with oatmeal and water. Continuous application may also be facilitated by the use of the *salve-muslins* and *plaster-muslins* introduced by Unna.

The most valuable applications are those of lead: ung. plumb. acet.; ung. plumbi glyc. subacet.; ung. plumbi subacet. compos. (B.P., 1867, liq. plumbi subacet. ℥vj, camph. gr. lx, white wax ℥viij, oil of almonds ℥xx); unguentum diachyli of Hebra (made like lead-plaster, with twice the quantity of oil); or solution of subacetate of lead ℥ss to vaseline or lard ℥j. Ointments of zinc oxide, oleate of zinc, calamine, and boric acid act similarly. Lassar's paste is zinc oxide and starch, each 24 parts, salicylic acid, 2 parts, and vaseline, 50 parts. In the more purulent or impetiginous cases mercurial ointments may be usefully combined with the preparations of lead or zinc; such as ung. hydrarg. ammon.; ung. hydrarg. oxidi rubri; ung. hydrarg. nitratis dil. The same substances can be used in lotions, such as the liq. plumbi subacetat. dil.; or a somewhat stronger one, 1 part each of liq. plumbi subacet. and glycerine to 30 parts of water; or lactate of lead made by shaking liq. plumbi subacet. ℥j with milk ℥ij; zinc oxide (℥ss with glycerine ℥ss to aq. rosæ ℥j), or calamine (gr. xv–xx to ℥j).

In some cases during the day, when lotions or ointments may be inconvenient, dusting powders may be employed, particularly oxide of zinc with an equal quantity of starch or French chalk, or boric acid mixed with four or five parts of the same substances.

In chronic cases where there is little discharge and much thickening and scaliness, more stimulant and irritating preparations may be employed, and especially those of tar, creosote, oil of cade, liquor carbonis detergens, and carbolic acid. They may be added to the other lotions or ointments just mentioned—*e.g.*, ung. picis liq. ℥ss or ℥j to ung. zinci or ung. plumbi ℥j; or liq. carbonis detergens ℥j to lot. plumbi or lot. calaminæ ℥vj; or they may be

used alone in inveterate and troublesome cases—*e.g.*, ung. picis liq. ℥ss or liq. carbonis detergens ʒss to aq. ℥vj. Ichthyol also gives good results as an ointment (ʒj to vaseline ℥j) or as a lotion (ʒj to ℥vj). For seborrhoic eczema, sulphur may be used unless the eczematous process is very pronounced: it may be added in the strength of from 3 to 10 per cent. to a zinc or boric ointment. Unna's ichthyol varnish is also of use: Ichthyol, 40 parts, starch 40 parts, albumen 1 to 1½ part, water to 100 parts.

Most of these methods of treatment will relieve the itching which accompanies the disease. Stronger sedatives are sometimes employed—for instance, cocain; but the exposed surfaces render such applications very unsafe.

The *internal* treatment of eczema must be directed to correcting every fault in the digestion or general health that may be detected. Gastro-intestinal troubles of children, and dyspepsia in others, must be treated by suitable remedies; delicate and anæmic patients should take iron, cod-liver oil, quinine, &c. If gout is a fact in the patient's history, laxative salines should be given; but it is not right to prescribe for gout unless there are some other indications than the eczema itself. Internal treatment may be called for to allay the terrible itching of some cases; for this purpose chloral and potassium bromide are most useful; the former especially in children—for instance, 2 or 3 grains in syrup to a child of six months given at bedtime and repeated near midnight; and the bromide in older people. Hyoscyamus, or tincture of hops, may be also given, and quinine seems to allay itching in eczema as well as in chronic urticaria.

Of specifics for eczema there are few. Modern practitioners appear to have little faith in arsenic, which was once universally used. It may certainly be tried in chronic cases, but it is less likely to be of service in acute forms. It should be given after meals in small doses gradually increased. M. Morris recommends antimony in small doses. Crocker thought he had success in inveterate cases with an indirect method of treatment—namely, counter-irritation over the neck or loins with mustard or blistering fluid, in order to influence the vasomotor system.

In acute eczema the diet should be that of a febrile case, chiefly milk and farinaceous food. In chronic cases very little alteration is required. Stimulants should be used in moderation only; and salt meats, as likely to increase thirst, may be well avoided. The patient should, of course, abstain from any food that obviously disturbs digestion.

The treatment of eczema in particular localities may be briefly mentioned.

Scalp.—Cut the hair short, remove crusts by oil or boric lint; apply weak iodoform ointment in pustular cases, or a mixed ointment of zinc oxide, lead acetate, and dilute mercuric nitrate ointment (ung. metallorum). In drier eczema, mixed with seborrhœa, use tarry applications—*e.g.*, liq. carbonis detergens.

Hairy parts of the face.—Cut the hair and shave as soon as it can be borne, then use astringent ointments; extract the hairs if pustules form round them.

Lips.—Mild astringent ointments of lead, zinc, or yellow oxide of mercury. The following is a good preparation: Acid. carbol. ℥ij; glycerini, ætheris, of each ℥j; sp. vini rect. ℥vj.

Palms.—After removal of crusts, apply the ointment spread upon strips of lint separately to each finger, and fit a kid glove over all. In chronic cases remove the thickened epidermis by the use of salicylic acid plaster or by soaking with pancreatic emulsion (Crocker), or papain (M. Morris). Salicylic acid or mercurial ointments may then be applied.

Nails.—Eczema of the nails is rare; they have a dirty, yellowish colour, and are pitted, grooved longitudinally, thickened, thinned, or split. Apply mercurial ointments, or those of salicylic acid, or of tar, wrapping up the ends of the fingers completely.

Legs.—This often results from varicose veins. Keep the patient in the recumbent position and raise the legs; or bandage carefully from foot to knee, or use Martin's rubber bandage. Apply astringent ointments or lotions.

PITYRIASIS

The name pityriasis (*πίτυρον*, bran) has been given to several affections in which the skin presents a bran-like appearance either from desquamation or undue accumulation of the horny layer of the epidermis. *Pityriasis capitis* and *P. circinata* appear to be due to bacilli or cocci; *P. versicolor* is due to a fungus, and is called tinea versicolor. *Pityriasis pilaris,* now called keratosis pilaris, and *P. rubra pilaris* are hypertrophic conditions of the epidermis about the hairs. In *P. rubra* the epidermic scales are often very large, and the disease is often called exfoliative dermatitis.

PITYRIASIS ROSEA

This is a superficial dermatitis accompanied by branny desquamation. It begins as a small spot on the trunk, neck, or arm, round or oval in shape, rose-coloured, and covered with fine scales. After a few days other spots appear, especially over the upper part of the trunk and neck, rose or reddish-fawn in colour, scaly on the surface, and slightly itching. The abdomen and limbs may afterwards be invaded. As each patch enlarges, the centre may fade, so that the appearance of rings is assumed. It generally subsides in the course of six or eight weeks. There is slight leucocytal infiltration of the corium, and proliferation of the epidermis. No organism has been proved to be its cause, and its origin is unknown. Sedative ointments of zinc or boric acid may be applied.

PITYRIASIS RUBRA

(*Exfoliative Dermatitis*)

This disease begins with a patch of erythematous redness on the chest, arm, or other part. It rapidly spreads over the whole body, either from the original patch or by the appearance of fresh patches which coalesce. The patches are bright red in colour, well defined at the margin, of no definite shape, and quickly become covered with large thin scales. The whole body may be thus affected in from two days to two or three weeks. The scales are small on the face, but larger on the trunk and limbs, detached at the margins, and frequently and abundantly shed, so that the bed is filled with dry, papery flakes, amounting to a pint or two in twenty-four hours. There is but little secretion from the skin, if any, and it does not discolour or stiffen linen. As a rule, there is little or no infiltration, and itching is not troublesome; but there may be some burning or tingling sensation. More infiltration occurs in old cases, and there may be exceptionally more itching, more secretion, and some fissures.

The disease may arise in those in perfect health, but it often follows eczema, psoriasis, erythema, or traumatic dermatitis. Acute cases are accompanied with fever, and its chronic persistence may induce ill-health, emaciation, and sometimes albuminuria. If recovery takes place there may be a relapse; but the disease is often persistent, and death occurs from marasmus, diarrhœa, pneumonia, or bronchitis. It occurs in both sexes and at all ages, but is comparatively rare in children.

Anatomy.—According to Crocker, it is a dermatitis, at first superficial, but afterwards involving the whole depth of the skin, and resulting in new connective tissue, cicatricial contraction of the same, pigmentation, hyperplasia of elastic fibre bundles, and obliteration of the papillæ, and of the sudariparous and sebaceous glands.

Diagnosis.—Exfoliative dermatitis has certain points of similarity with eczema, psoriasis, and pemphigus foliaceus. From the first it is distinguished by the extent of body involved, by the absence of secretion, and by papery scales instead of yellow crusts; from *psoriasis* by the extent, by the absence of infiltration, and by the scales not being massed into thick flakes; from *pemphigus foliaceus* by the absence of bullæ with discharges preceding the scales. From *lichen ruber*, also, it is distinguished by the absence of papules and of infiltration. Uræmic dermatitis sometimes closely resembles this disease; and Savill recorded a number of cases of epidemic dermatitis occurring in a Poor-Law infirmary, of which many were very like dermatitis exfoliativa, and others like eczema.

Treatment.—Emollient applications are mostly recommended, such as olive oil, linimentum calcis with zinc oxide and calamine, lead and zinc ointments, glycerine of lead subacetate, or lactate

of lead; or weak tarry preparations, such as carbolic oil, or liq. carbonis detergens in vaseline, or ichthyol soap. But stronger tar preparations may be too irritating. The applications must be frequently made over a long period. Internally, tonics, quinine, and nutritious diet must be given, and in older cases arsenic is of value.

PSORIASIS

This disease consists in the formation of raised red patches covered with thick, silvery white adherent scales. In a great number of instances the lesions appear first on the knee over the patella, ligamentum patellæ and tubercle of the tibia, and on the elbow over the olecranon. It begins with papules, which enlarge into large flat plaques; quite early the papule is seen to be covered with an opaque scale, and with its enlargement in size, the scale becomes thicker, especially in the centre, and silvery white in appearance. The scale is rather firmly adherent, and co-extensive with the red plaque, so that the red colour can often only be seen at the edge. If the scale is removed it leaves a shining, moist-looking, but actually dry bright red surface, in which examination with a lens will show a number of deeper red points, the hyperæmic papillæ. The patches are at first roundish or circular, and enlarge to half an inch, an inch, or more in diameter; fresh patches come out near the first or in other parts of the body. If a patch becomes very large it may heal in the centre, and thus form a ring; coalescence with other rings will produce serpiginous or gyrate figures. The patches may spread sufficiently to cover large areas of the body continuously, so that the original shape of the spot cannot be detected. The old names given to indicate these different stages have little more than a descriptive value, such as *P. punctata, P. guttata, P. nummularis, P. circinata, P. gyrata, P. diffusa, P. universalis.*

Next to the knees and elbows, the adjacent extensor surfaces of the leg and forearm are most commonly affected, and then the thighs, back, loins, chest, and abdomen; and in all regions a very striking symmetry is observed. The face and scalp are not often attacked, and the palms and soles rarely. The nails are not infrequently involved: they become variously altered, opaque, thickened, pitted, furrowed transversely; or immensely thickened, and discoloured.

The amount of scale varies in different instances or in the same case at different times. In *P. rupioides* the scales are heaped into small conical masses, each on its circular base.

The eruption is always dry, never moist or scabbing as in eczema. Itching is variable, but not, as a rule, severe. The general health is often perfectly good, or even robust.

The disease breaks out spontaneously, often in early childhood, and even if not treated subsides after three or four months, to recur again after a quite uncertain interval. Sometimes the recurrence

is twice a year ("spring and fall," as it is often expressed), or a period of years may intervene. In other cases a slight amount of eruption persists, and extensions take place from time to time. During recovery, pigment stains mark the situation of the patches especially after the use of arsenic.

Ætiology.—It affects both sexes, and nearly all ages; but it more commonly begins in early life. The only other certain fact in its causation is that it is hereditary; the view that gout and scrofula are causative antecedents has little in favour of it.

Pathology.—It is an inflammation of the papillæ and corium, with increase of the stratum mucosum, down-growth of the same between the papillæ, which appear correspondingly enlarged, and increase of the horny layers (*keratosis*). The silvery appearance of the scales is due to the inclusion of minute air globules. The eosinophile leucocytes in the blood are often increased.

Diagnosis.—In most cases it cannot be mistaken. Patches of dry *eczema* may resemble it, but the edges are not so sharp and the scales are not so thick and silvery. In psoriasis of the scalp the scales are often yellow, and look like impetigo crusts; but psoriasis spreads beyond the scalp on to the forehead or neck, and there is always psoriasis of some other parts of the body which will be distinctive. These last two points will also serve as between psoriasis and *seborrhoic eczema*. *Lichen ruber* and *pityriasis rubra* must be distinguished by the descriptions given. Patches of *tinea circinata* may look like psoriasis, but their small number, want of symmetry, small scales, and the results of microscopic examination will show their nature. *Lupus erythematosus* is recognised by its position on the face, the greater thickening of the skin, the sebaceous plugs and the scars; and *scaly syphilides* by the small size of the lesion, the slight scaliness, the browner colour, and the concomitant symptoms.

Treatment.—The best treatment is a combination of internal and external remedies, but one of these alone will suffice in some cases. Internally for acute cases salicin was recommended by Crocker; it may be given in doses of 15 or 20 grains three times a day. For chronic cases arsenic is most frequently successful; it may be given in increasing doses of the liquor, beginning with 3 or 5 minims, and stopping at 10 or 15 minims, always after meals. Other forms in corresponding doses, arsenious acid in pills, liq. arsen. hydrochl., or liq. sod. arseniatis, may be equally well employed. Some other drugs are sometimes of use: carbolic acid in $\frac{1}{2}$-grain doses (Kaposi); turpentine in 10 to 30 minim doses (Crocker), vinum antimoniale in 5 or 10 minim doses (M. Morris), sodium salicylate, and iodide of potassium. The last requires to be given in very large quantities, and cannot always be borne.

The best *external* treatment is that by tar and allied preparations: ung. picis liquidæ, creosote ointment, ointment of cade oil (℥j to ℥iv to ℥j), or liq. carbonis detergens (℥ij to ℥j). The scales should be removed first by the use of alkaline baths, the wet pack, soft

soap, or vaseline. The selected preparation should then be both rubbed into the part and left in contact, the limbs being enveloped in old flannels to prevent staining of the clothes. Besides the tarry preparations just mentioned some others are efficacious : chrysarobin ointment, which must be used with caution, as it often sets up a dermatitis beyond the limits of its application, and stains the hair and clothes of an orange or golden colour ; pyrogallic acid (℥ss or ℥j to ℥j of benzoated lard), which must be used over a small area at a time ; ointment of salicylic acid (5 to 10 per cent.), also for small areas ; and resorcin (gr. x or xx to ℥j of lard). Crocker also recommended turpentine or oleum pini sylvestris, alone or diluted with olive oil.

Patients who are not in good health, but suffer from anæmia or strumous delicacy, should be treated with iron, cod-liver oil, or quinine, which need not interfere with the specific arsenical course.

LICHEN

This term has been long in use to signify any sort of papular eruption, but is now often restricted to one disease, *Lichen ruber planus* or *Lichen planus*.

The conditions formerly called *Lichen simplex* and *L. agrius* are papular forms of eczema; *L. lividus* is purpura round the hair-follicles ; *L. urticatus* is urticaria in children ; *L. circumscriptus vel circinatus* is pityriasis circinata or seborrhœa corporis ; *L. pilaris* is now called keratosis pilaris ; *Lichen ruber acuminatus* is pityriasis rubra pilaris ; and *L. scrofulosorum* is regarded as a tubercular lesion, and will be described under Tuberculosis of the Skin.

LICHEN PLANUS

L. planus consists of raised flat patches of a dull red or almost violet colour, and a surface smooth, shining, or covered with quite small scales. The patches arise from the aggregation of papules, which are at first discrete, and then become continuous by the growth of fresh papules in the intervening spaces. The papules are flat, shining, square or polygonal, and sometimes with a minute depression in the centre. Wickham pointed out that white opalescent points or striæ forming a fine network are visible under a lens, especially if the surface has been moistened with oil or water. The eruption is more or less symmetrical, and appears first on the wrists and forearms, and on the inner side of the knees, and then on the extensor surface of the arms or legs, the ankle, foot, the flank, hip, and lower part of the abdomen. It also tends to appear on parts subject to pressure—for instance, the waist, the legs where the garter presses, and the palms and soles ; but in these last two situations there is only a general thickening of the epidermis, with white spots where the horny layer is cracking. Sometimes the

papules have a linear distribution along the course of the nerves. The rash on the skin is often associated with white spots on the tongue and inner side of the cheeks. The head and face are said never to be affected. There is only moderate itching, and the health is influenced only in proportion to the extent of skin involved. In generalised extensive disease marasmus and death may ensue. When the lesions subside they leave a very persistent stain. On the lower extremities, and in connection with varicose veins, the papules may grow to a great size (*L. hypertrophicus*).

Ætiology.—In many cases no cause can be discovered; in others worry, anxiety, insufficiency of food or defective digestion have preceded the disease. It is most common between the ages of twenty and fifty, and rarely attacks children.

Pathology.—The inflammatory process in L. planus begins round a sweat duct in the upper part of the corium; there is a dense growth of connective-tissue cells, according to some, of granulomatous type. This is followed by increase of the cells of the stratum mucosum, and thickening of the horny layer. There is irregular thickening of the eleidine, which causes the appearance of white striæ, above mentioned.

Treatment.—The treatment is not unlike that of psoriasis. Internally arsenic should be given steadily in full or increasing doses for a considerable time; it is most successful in chronic cases and less certain in acute; perchloride of mercury and salicin are also recommended. The local treatment consists in the use of tarry preparations, such as ung. picis liq., ung. creosoti, liq. picis carbonis, thymol, and carbolic acid. If there is much hyperæmia more soothing applications, such as lead or zinc lotions, may be desirable for a time. The general health also requires attention; bodily and mental rest, nutritious food, the usual tonics, and, perhaps, change of air.

PARAKERATOSIS VARIEGATA

This is a condition somewhat resembling a lichen, consisting of small flat papules, covered each with a fine adherent scale, which can be scratched off without bleeding. When the scale is removed the papules are yellowish red on the body or bluish red on the extremities. The papules run together in such a way as to include healthy areas of skin in a sort of meshwork, and thus give a retiform or marbled appearance to the skin. The whole of the surface may be affected, except the face, scalp, palms, and soles. It occurs especially in otherwise healthy adult males. It is chronic in its course, but subject to remissions and exacerbations; unaccompanied by subjective symptoms, and very resistant to treatment. Histologically, it is an inflammation of the subepidermal layer of the cutis, with dilated vessels, œdema, and cell-infiltration; as well as some œdema and thickening of the epidermis.

PRURIGO

This is a papular disease accompanied with severe itching (*prurio*, I itch). It must be distinguished from *pruritus*, which means simply the sensation of itching, and not structural change. Thus, we have pruritus in prurigo itself, but also in eczema, in jaundice, and in other conditions. One disease that is often described as prurigo (*P. senilis*, *P. pedicularis*, *phtheiriasis*) is obviously the result of the irritation of the body louse, and I shall speak of it when treating of parasites. Prurigo consists originally of minute papules in the skin, which are at first not so much visible as palpable, having the colour of the skin, and only later becoming pink and red. They are not collected in groups, but scattered. As they are accompanied by severe itching, they are soon scratched, the heads of the papules are removed, and a small blood-scab is the result. More violent scratching leads to enlargement of the papillæ, thickening and roughening of the skin, the natural furrows are deepened, the surface is covered with mealy scales, the downy hair is destroyed, and when the hand is passed over the skin it feels like a nail-brush or like rough brown paper. Besides the excoriation of the papillæ, more extensive scratch-marks, abrasions, and scars may be caused, and other secondary lesions, such as eczematous patches, urticaria, pustules, enlargement of the femoral, axillary, or elbow glands, and, finally, more or less deep pigmentation of the skin.

The parts first affected are the extensor surfaces of the legs and arms, especially the former. The chest, back and front, the abdomen, and gluteal regions are all affected; even a few papules may appear on the face; but the flexures of the elbow and knee, the axillæ, the genitals, the ankles, wrists, palms, and soles are always spared.

Ætiology.—It is more common in males than in females, and among the poorer classes. Different statements are made as to the influence of cold, but it is certain that winter cold does cause in some people a pruriginous condition, especially of the legs (*P. hyemalis*). It commonly begins in infancy, and has sometimes been preceded by an urticaria; it continues, unless vigorously treated, for the rest of life.

Varieties.—*P. mitis* and *P. ferox* have been described: they appear to differ only in intensity. Hebra regarded as a special and incurable form the very intense cases which he saw in Vienna; but there seem to be cases intermediate between those and the milder forms commonly seen in England.

Pathology.—The early anatomical change is an exudation into the papilla of leucocytes or serum, the former collecting about the vessels: there is also infiltration and imperfect vesicular formation in the epidermis. Most writers believe that these anatomical changes are secondary to itching and irritation.

Diagnosis.—Very similar conditions are produced by pediculi,

and in a doubtful case these insects should be looked for (*see* Phtheiriasis). Prurigo is, however, distinguished by the age of the patient, the long history of the disease, and the distribution of the lesions.

Treatment.—This must consist of the frequent use of warm water, the Turkish bath, or alkaline baths, and the thorough inunction of soap and emollient ointments. Tar and sulphur preparations are also of value. Whatever method is employed must be continued daily and perseveringly. The following may be used: Spermaceti ointment alone or with the addition of cod-liver oil, or tar ointment; equal parts of soft soap and spirit, or a fluid glycerine soap; sulphur ointment, or Vlemingkx's solution (containing sulphides of calcium); carbolic acid (2–5 grains to aq. 1 ounce); menthol (5–10 grains in dilute alcohol 1 ounce, or as a soap); solution of coal-tar (1 drachm to 8 ounces); or tar baths (brushing the surface all over with tar, and then remaining in a hot bath for three or four hours). Internally help may be obtained from arsenic, carbolic acid, cannabis indica, and antipyrin. Nutritious food, cod-liver oil, wine, &c., should also be given.

DISEASES OF THE SKIN DUE TO MICRO-ORGANISMS

Infective diseases of the skin have more than once been referred to in connection with the general infectious diseases. Thus erysipelas is a streptococcal infection, boils and carbuncles are due to staphylococci, and the skin lesions of leprosy, frambœsia, syphilis, glanders, and diphtheria are due to their respective micro-organisms.

IMPETIGO

It has already been said that the infection of eczematous surfaces with pyogenic organisms causes a change in them which is called impetiginous. Impetigo then is a pus-producing inflammation of the surface of the skin, and the term was formerly applied to many pustular eruptions; but it is now almost limited to one on the scalp, and to an eruption which has been separately described as *Impetigo contagiosa.*

I. contagiosa occurs as flat vesicles, enlarging into pustules from a quarter to half an inch in diameter, with scarcely any surrounding inflammation. They occur especially on the face, about the mouth, nose, and chin, and the occipital region. They are generally few in number and discrete; and they appear to cause similar lesions by contact in other parts of the skin, or in other persons. In children, the head louse, pediculus capitis, is the commonest

determining cause of *Impetigo capitis*. The insects cause itching, scratching follows, and the lesions thus produced give the opportunity for infection. Pyogenic organisms, streptococci especially, are present. A contagious impetigo occurs also among football players at schools (*football impetigo; scrum-pox*); and staphylococci have been found in the pus.

Impetigo of the scalp forms thick crusts of yellowish or greenish-yellow colour, matting the hairs together; it is most marked in the occipital region when due to pediculi. The suboccipital glands are generally enlarged and may suppurate.

Treatment.—Pediculi should be sought for and destroyed (*see* Phtheiriasis). The local lesions of impetigo may be treated by removal of the scabs and the use of a dilute white precipitate ointment (5 grains to the ounce) or the mixed ointments of zinc, lead, and mercury. Tonics, such as cod-liver oil and iron wine, are often desirable at the same time.

ECTHYMA

This is a similar suppurative lesion, due to streptococci, consisting of scattered pustules with resulting scabs on the trunk and extremities, and occurring in cachectic and anæmic persons. The lesion is deeper seated than impetigo and extends to the corium, there is more red areola around the pustule and scab, and scarring often occurs. The general health must be treated, and the same local applications may be used as in impetigo.

PITYRIASIS CAPITIS

(*Dandriff. Scurf*)

This is common in children, but continues in later life. It consists of a large number of minute whitish branny particles, which develop over the whole head, are readily detached, and lie loose among the hair, or fall on to the shoulders. If it continues for long it may lead to a weak growth of hair, or actual falling out. It may be accompanied by a slight redness of the scalp of the affected part.

This condition has also been regarded as being due to excessive secretion of sebum, a *seborrhœa sicca;* but according to Sabouraud the sebaceous follicles are not affected; and there are constantly found in the scales organisms, the bottle-bacilli of Unna, or spores of Malassez, to which the excessive formation of epidermic scales may be due. In more greasy scales there is in addition the staphylococcus epidermidis albus (morococcus of Unna).

Treatment.—The head should be shampooed from time to time: and mild antiseptic applications should be made daily; as, for instance, lotions containing salicylic acid, 15 or 20 grains to the ounce of water, or perchloride of mercury, 1 in 2000; or ointments of

zinc oxide and oleate, mercurial ointments, or carbolic oil; or an ointment containing 15 grains of sulphur and 10 of salicylic acid to the ounce, or resorcin and sulphur, of each 20 grains to the ounce.

PITYRIASIS CIRCINATA

(*Lichen circumscriptus. Seborrhœa corporis*)

This is constantly associated with pityriasis capitis, and similar organisms are found in the scales. It is more frequent in men than in women; and the patients are found to wear flannel shirts or vests. The eruption consists of small flat dusky red papules, combining to produce circular patches a quarter or three-eighths of an inch in diameter, which may clear in the centre, and ultimately form rings of a larger size. By running together they result in scalloped and gyrate figures. The papules are often covered with a yellowish-brown scale. They occur almost exclusively over the sternum and on the back between the shoulders, but may extend from the middle line in front over the pectoral regions. The eruption itches slightly, but otherwise causes little discomfort, and may be only noticed when the patient consults his doctor for some other complaint. The skin is often greasy.

Diagnosis.—The situation, the annular shape, and the frequent yellowish tinge are characteristic. Only if very extensively developed in a gyrate form could it be suspected of having a *syphilitic* origin.

Treatment.—Pityriasis circinata is cured speedily by tar or creosote ointment, and glycerine of borax or thymol, and preparations containing sulphur and salicylic acid, such as 30 grains of sulphur and 10 grains of the acid to an ounce of vaseline; but it readily returns if the local conditions are not altered by frequent washing, and suitable changes of underclothing.

TUBERCULOSIS

Tubercle affects the skin in several ways; and the following lesions and diseases are now known to be the results of its invasion: *Miliary tuberculosis, acute and chronic ulcers, Scrofulodermia, Lupus vulgaris, Lichen scrofulosorum, Verruca necrogenica,* and *Erythema induratum.* Miliary tubercles of the skin are rare, and only occur as a part of a general miliary tuberculosis.

TUBERCULAR ULCERS

These may be acute or chronic. The former occur rarely except in patients suffering from visceral tubercular disease. They first appear as dull, red swellings, which break down into ulcers with thin undermined edges. They are little likely to heal in view of the internal disease. Chronic ulcers may be scattered over different parts of the body; they are round or oval, with pale œdematous

granulations, and somewhat undermined edges. Mildly stimulating applications externally, cod-liver oil and quinine internally, and the best hygienic conditions, are required. For both kinds of ulcers the Röntgen rays may be applied.

SCROFULODERMIA

This consists of tubercular abscesses and ulceration of the skin, often originating in tuberculosis and caseation of subjacent glands. Sinuses form in connection with the suppurating glands, and the skin is infected from them. It occurs in the neck and groin and other parts. The skin becomes red and inflamed over a swollen gland, and ultimately gives way, forming an ulcer with undermined edges. The process may extend over an area of some inches, and ultimately cure may take place, with the formation of irregular scars, with fibrous bands, and scattered pigmentation.

Surgical treatment, curetting, the Röntgen rays, and general hygiene are the means required to bring about healing of the lesions.

LUPUS VULGARIS

This consists of superficial infiltration of the skin, which spreads slowly, while healing by cicatrisation in the older parts, with or without preceding ulceration.

It occurs in both sexes, and is more common in young people; indeed, it more frequently begins before the age of thirty, and its progress is likely to be less rapid after that age. It is more common among the poor than among the wealthy. In spite of its apparent histological identity with tubercle, it is not hereditary, and the subjects of lupus rarely suffer from the typical tubercular lesions.

It begins usually as a small pink or brownish-red spot, and gradually enlarges; then small nodules are felt in it, and it becomes slightly raised above the surface. It is rather sharply outlined, smooth on the surface, with a somewhat translucent look, and as it enlarges it may have a more yellow or orange colour. The nodules consist of a vascular granulation tissue—that is, closely aggregated small cells, with numerous capillaries; and the intervening tissues of the cutis and the papillæ are infiltrated with leucocytes, and strings of cells form in the course of the lymphatics. In the centre are giant-cells and tubercle-bacilli.

There is neither pain nor itching. The growth of the disease is slow; it advances irregularly at its edge, and the surface may be more or less scaly; but it ultimately undergoes one of two changes. Either it cicatrises directly, by some of the cells forming fibrous tissue, and others, together with the normal tissues of the skin, become absorbed; or the disease proceeds to the surface, the epidermis is involved, the cells degenerate and break down, and an ulcer is formed, which is covered with purulent crusts and scabs. In such an ulcer the edges are raised, the lupus nodules can be

recognised in the base, and the pus is thin and scanty. In course of time a lupus patch presents a somewhat irregular arrangement of fresh lupus tissue, of crusted ulcers, and thin, white, ill-defined cicatrices. The patch is generally single and unsymmetrical.

The part most commonly first invaded is the face, especially the ala of the nose, the edge of the lip, the cheek or the eyelid; or it attacks the ear or the neck. It is much less common on the trunk or limbs, and rare on the scalp. The mucous membranes of the nose, mouth, lips, gums, hard and soft palate, epiglottis, and larynx are sometimes affected.

Though lupus destroys the skin in which it grows, it is only indirectly destructive of other parts—that is by pressure and atrophy. Thus, if the disease spreads over the face and nose, in course of time the resulting scars will contract, the lower eyelids are everted, the gums are exposed, the tightly stretched skin compresses and atrophies the cartilages of the nose, and much deformity results. These effects were at one time attributed to actual invasion of the cartilage by the lupus, and its subsequent destruction by ulceration, and so a division into *Lupus exedens* and *Lupus non-exedens* was made. But it is probable that lupus, if it sometimes grows into the subcutaneous tissues, rarely involves fasciæ and cartilages, and never bones, muscles, other deeper structures, or the internal organs. Some cases of *L. exedens* were no doubt tertiary syphilis or rodent ulcer.

In spite of the persistence of the disease, the patients are mostly in very good general health.

Diagnosis.—It is distinguished from all ordinary cutaneous affections by the red infiltration of the skin, accompanied by cicatrisation. It is most likely to be confounded with cancer, rodent ulcer, or tertiary syphilitic ulceration. The diagnosis is most important as between syphilis and lupus; in the former, the edges of the ulcers are not tuberculated, and the skin around is often deeply pigmented, the lesions are not so extensive, but ulcerate more deeply, and often a gumma or deep suppuration precedes the breach of surface; the lesions of syphilis may be multiple, or accompanied by disease of other parts of the body, such as periosteal nodes, lardaceous viscera, &c.

Treatment.—Local treatment is the only kind that can be with certainty efficient. Cod-liver oil and tonics may assist in some cases, and the use of *tuberculin*, in association with *opsonic tests*, may be of value, especially where the lesions are extensive and constantly recur. The local methods are *excision*, the *cautery*, *caustics*, *scraping* with a metal edge (sharp spoon), *scarification*, *Finsen's light treatment*, and the use of the *Röntgen rays*. The first four cannot always be used for lesions on the face or neck, when the appearance of the scar may be of importance. The principle of the scraping treatment is that the lupus tissue is less resistant than the healthy tissue beyond it. The patient is anæsthetised, and with the instrument all lupus growth is forcibly removed,

while the firm healthy skin is left. With lunar caustic a similar result may be effected; it will break down and destroy more readily the infiltrated skin than the healthy, but it is more likely to want repeating than a well-conducted scraping. Crusts and scabs must of course be first removed, and the new wound may be dressed with zinc ointment. Caustic potash, arsenical paste, and acid nitrate of mercury have also been used, but are very painful, and require careful management to prevent injuring the sound skin. Scarification is performed by making hundreds of punctures into the affected skin, close together and about two lines in depth; and this operation is often repeated. By this means vessels are divided and obliterated, and the new growth is more or less starved. It is obviously more suited to parts that are not ulcerated. In some cases ointments of salicylic acid, ichthyol, pyrogallic acid, or calomel render good service.

Many cases are now being benefited by the Röntgen rays or by Finsen's light treatment. In the latter, both sunlight and the electric arc light can be used, the heat rays in either case being as far as possible obstructed, while the blue and violet rays are concentrated on the spot; at the point of application the skin is compressed so as to diminish the quantity of blood in it. The applications are made for an hour daily, and require to be continued for weeks or months.

LICHEN SCROFULOSORUM

An eruption consisting of small papules, at first red or pink, later fawn-coloured or almost yellow, arranged in roundish groups, or circles, or segments of circles. On the older papules a minute scab is formed, and after a time the papules subside and leave only a yellowish pigmentation. They occur on the trunk, especially at the sides, and rarely on the limbs; the occurrence on the limbs is more frequent in children than in adults. Itching is absent or very slight. The disease progresses by the appearance of fresh crops of papules from time to time, so that it may last for months or years.

Many patients have enlarged lymphatic glands, caries, or other bone lesions, or ulceration of the skin; but phthisis is not common. It occurs in both sexes, but has been seen more often in males. It is most common in children, and rare after early adult age.

The papules have a characteristic tubercular structure with epithelioid cells and giant cells; but the tubercle-bacillus cannot always be demonstrated.

Treatment.—Cod-liver oil has been used internally; and externally cod-liver oil, vaseline, vaseline with liq. plumb. subacet., calamine lotion, thymol, and oil of cade.

VERRUCA NECROGENICA

This is an indurated warty growth or thickening occurring on the hands, knuckles, and rarely the arms of persons exposed to post-mortem infection, whether animal or human (butchers, medical men and students, post-mortem assistants). It begins as a dusky red indurated patch of skin, which will yield some sero-pus on pressure; and around the unhealthy granulation there is papillary hypertrophy and epidermic thickening. The lesion is very persistent, and may last months or years. It has been shown in many cases to be tubercular.

Treatment.—As a small localised lesion its destruction by caustics or electric cautery may be the best; otherwise salicylic acid should be applied as ointment (2 per cent.) or as plaster, or dissolved in collodion (℥j to ʒj) or in alcohol (saturated); and tuberculin injections may be employed.

ERYTHEMA INDURATUM

This form of cutaneous tubercle occurs most often in adult females in feeble health, and affects especially the lower outer or back part of the legs. Deep-seated indurations, the size of peas or nuts, form under the skin; at first they can only be felt, but afterwards encroach on the skin, and cause dusky red or purplish projections. They may disappear, and reappear from time to time, or they break down into ulcers with surrounding œdema. They may be mistaken for gummata, and exceptionally they may be independent of tubercle, as shown by the failure of the tuberculin-test and the absence of bacilli (Whitfield).

Treatment.—Prolonged rest, elevation of the limb, and careful bandaging promote recovery.

RHINOSCLEROMA

This is a dense infiltration of the septum and alæ of the nose, rendering it as hard as ivory, thick, and rigid. The surface is smooth or irregular, the colour normal or brownish-red; the mucous membrane is affected as well, and the orifices may be blocked by its swelling. Not infrequently the adjacent upper lip, and sometimes the cheeks, are involved. The changes are a dense infiltration of the corium and papillæ with plasma cells, the presence of large translucent degenerated cells (Mikulicz cells), and bacilli, the bacilli of Frisch, which have a close resemblance to the pneumonia-bacilli of Friedländer, and are found chiefly in the Mikulicz cells, but also in the plasma cells and tissue. Removal by the knife or destruction by caustics is necessary for cure.

NEW GROWTHS IN THE SKIN

Only a small number of these will be here described. For *nævi*, or vascular tumours, *carcinoma*, *epithelioma*, *rodent ulcer*, and *sarcoma* the reader is referred to works on surgery.

FIBROMA MOLLUSCUM

This is a soft, flaccid, wrinkled, often pendulous tumour, consisting of a covering of scarcely altered cutis and epidermis, containing a fibrous meshwork with a variable proportion of round cells and albuminous fluid. The tumours may be very few, or exceedingly numerous; they vary in size from a pin's head to the head of a man, and they occur especially on the trunk. They may apparently be congenital, but are generally first seen in early childhood. According to von Recklinghausen, they are really neuro-fibromata, starting from the fibrous sheaths of the smaller cutaneous nerves, and thence invading the fibrous structures of the vessels, the sweat-glands, and the hair-follicles. In an allied condition there are no separate tumours, but the skin is thickened and overgrown, or lies in large, loose, overlapping folds (*dermatolysis*). Cases of *multiple fibromata* are rare; they are known as *Recklinghausen's disease*.

Treatment.—Removal by the knife is the only possible treatment; but if the tumours are very numerous, only such growths can be removed as are in specially inconvenient positions.

MOLLUSCUM CONTAGIOSUM

This name is given to small tumours on the skin, which are from one-tenth to a third or half an inch in diameter, lenticular or hemispherical in shape, occasionally globular or pedunculated, somewhat irregular or nodulated on the surface, and of a yellowish-white colour. In the smaller tumours there is often a minute opening in the centre—in the larger there are several; and if the tumour be firmly squeezed, a little milky juice exudes from these apertures. Examined under the microscope, the juice is seen to consist of minute oval glistening bodies, the *molluscum corpuscles* or *bodies*. A vertical section through the tumour shows it to have a structure somewhat like that of a racemose gland. There are lobules separated by fibrous tissue: each lobule has, externally, a row of columnar cells, within this are more oval epidermic cells, and in the centre of each lobule is a collection of the glistening opaque molluscum bodies. The lobules in the larger tumours do not converge to a central duct or opening, but rather lie side by side, and open separately upon the surface.

The process appears to begin by a conversion of the prickle-cells of the rete Malpighi into the molluscum bodies, which are now stated to consist of keratin. The adjacent part of the rete enlarges downwards into the corium, and the septa between the apparent lobules are the fibrous remains of the papillæ. There is no necessary connection with sebaceous glands or hair-follicles. No micro-organism has been identified.

The growths occur in children and adults; and are situate on the face, arms, or hands, the mammæ of women, and the genitals of men. They have been regarded as contagious, from the fact of their appearing in members of the same family, in mother and baby at the breast, and so on; but it is very difficult to produce them by direct inoculation. They may remain unchanged for long periods, they may suppurate, or they may subside and disappear.

Treatment.—This consists in pressing out the contents, either directly or after incision, and applying a little caustic; or by removal with knife or scissors.

CHELOID

Cheloid is a growth of the skin and subcutaneous tissue, consisting chiefly of dense bands of fibrous tissue, containing in its earlier stages numerous spindle-cells. Its more common seats are on the chest, over the sternum, on the mammæ, on the neck, back, lobules of the ears, and on the limbs. It is usually single. It begins as a flat, smooth, pink nodule, which extends laterally to a considerable size, and becomes paler in the centre, while the skin around is more or less reddened. After a time, bands and ridges, separated by furrows, develop, running in various directions across the tumour and into the surrounding skin. By the slow contraction of these bands, much deformity may be caused, and movements of adjacent joints may be seriously restricted. The growth of the tumour is often accompanied by considerable pain and tenderness.

Similar growths not infrequently develop on former scars, such as those of cuts, burns, acne, varicella, vaccination, or small-pox. These have been called false cheloid, but it does not seem that they are essentially different. Cheloid grows slowly, and rarely disappears spontaneously. If removed by a knife or caustic it almost inevitably returns; but it always remains a strictly local disease, invading neither lymph-glands nor viscera.

Treatment.—Cure has been obtained by the use of Röntgen rays; and by subcutaneous injections of fibrolysin and of thiosinamin. The latter is used in a 10 per cent. solution in diluted glycerine, of which 5 to 10 minims may be injected daily. The severe pain of cheloid may demand local or general anodynes.

MYOMA. NEUROMA. LYMPHANGEIOMA

These occasionally occur as cutaneous affections.

Myoma occurs in rare cases as multiple small, hard nodules from the size of a pin's head to that of a pea or bean, on the face, trunk, or limbs. Each is a small tumour in the corium, consisting of smooth muscular fibres (*leiomyoma*), related apparently in some cases to the arrectores pilorum.

Neuroma forms multiple, painful small growths in the course of the nerve-fibres of both trunk and limbs (*see* p. 221).

Lymphangeioma is a rare growth, due to dilatation of lymphatic vessels into visible cysts, and overgrowth of the intervening connective tissue. It has been seen in association with ordinary vascular nævi.

XANTHOMA

(*Xanthelasma. Vitiligoidea*)

In the most common variety (*X. planum*), one finds generally on each upper eyelid, near the inner canthus, a small sharply-defined patch of whitish-yellow, soft, smooth skin, level with, or scarcely raised above the general surface. Such patches may remain stationary for years, or may slowly increase, spreading outwards along each upper lid; or other patches appear on the lower lids, and the orbit is completely surrounded by a broad patch of the altered skin. In many cases no other part of the body is affected; but in others spots and streaks of a similar kind appear on the trunk, on the backs and palms of the hands, and the soles of the feet, or on the scrotum. They have also been seen in the mucous membrane of the gums, palate, side of the tongue, larynx and trachea, and in the mucous lining of the bile-ducts.

Another form (*X. tuberosum*) consists of firm, rounded nodules from the size of a pea to that of a nut, occurring on the skin over the elbow, on the knuckles, and on the lobules of the ears. These growths occur, as a rule, in persons of middle age or older, and are more frequent in women than in men.

The most extensive lesions in the above forms are associated with long-standing jaundice, but small patches affecting the eyelids alone are often seen without jaundice, and are thought by Hutchinson to be related to attacks of sick headache.

Xanthoma diabeticorum is a rare form which occurs in those who have glycosuria. It appears as yellow conical spots surrounded by a red raised area: these are seen first on the extensor surfaces of the arms, and at the lower part of the back and abdomen; and subsequently in other parts. They often subside rapidly.

Anatomy.—In the flat patches the corium shows a newly-formed connective tissue, with large round or fusiform cells, often multinucleated, filled with fat granules and closely aggregated fat drops.

The nodules have a similar structure, but the fibrous tissue is more abundant. In a very rare form (*Xanthoma of Balzer*) the chief lesion is thickening and deformity of the elastic fibres.

Treatment.—Small patches may be excised, but the removal of larger patches from the eyelids would risk serious deformities. If their removal is called for, caustics are to be preferred. In other cases the galvano-cautery at a dull red heat, or the Röntgen rays, may be employed.

MYCOSIS FUNGOIDES

In this rare disease, the skin is affected by a number of tumours, the appearance of which is often preceded, sometimes for years, by erythematous, eczematous, or urticarious patches. The tumours vary from the size of a bean to that of an orange, or larger. They are round, oval, or lobulated, the skin is stretched over them, tense and shining; the skin around them is often infiltrated. After a long duration some of them may shrink and disappear, others ulcerate on the surface and form fungating masses, discharging a clear watery serum. In this stage they are generally painless and free from itching or smarting. The disease lasts months or years, but ultimately the health fails, and the results have often been fatal. The view has been held that the disease is an infective granuloma, rather than a sarcoma, or a lympho-sarcoma; but no organisms have been found; and the tumours consist of lymphoid cells in a fine stroma of connective tissue.

Treatment.—Great improvement, though sometimes only temporary, has been recorded from the use of Röntgen rays.

HYPERTROPHIES OF THE SKIN

CALLOSITIES AND CORNS

These are produced by friction and pressure.

A *callosity* consists of a hypertrophy of the horny layers of the epidermis, and is familiar on the ball of the great toe, the heel, the hands of the working man, of oarsmen and others, the tips of the fingers of those who play the violin, &c.

The *corn* (clavus) is a local thickening of the epidermis resulting in a conical downgrowth, which presses upon the subjacent papillæ, causes their atrophy, and sets up inflammation and hypertrophy in the surrounding papillæ. Corns are common, as is well known, on the toes, especially the outer side of the little toe, the dorsum and the sides of the other toes. The pain of the ordinary corn is largely due to the little plug being driven down on the cutis beneath, but spontaneous shooting pain is often present. When the corn lies between two toes and is kept constantly moist, the thickening is less

marked; but the inflammation is more obvious, and the part is often extremely tender (*soft corn*). Occasionally corns will inflame and ulcerate, or a cyst or bursa forms under the corn, constituting a *bunion.*

Treatment.—Corns may be cured, and almost entirely prevented, by the use of properly shaped boots. The sole should be as large as, or slightly larger than, the sole of the foot as it shapes itself in the standing position with the weight of the body upon it. If the boot sole is narrower than the sole of the foot, the upper leather will be in close contact with the edge of the foot in any movement, and constant friction will be the result. The inner edge of the sole should be straight, and pointed boots should be strictly avoided. If corns have formed, they must be treated by soaking in hot water, and shaving with a sharp knife or razor, when the dry white plug will be met with and can be removed. A corn plaster may then be worn, or the toe may be simply strapped with a good linen plaster, by which, with properly constructed boots, the friction will be reduced to a minimum. Soft corns may also be carefully shaved, and pressure removed by cotton-wool between the toes, or by a turn or two of narrow strapping below the corn. The thickened epidermis may also be removed by the application of salicylic acid, either as a 2 per cent. ointment, or as plaster, or in solution in collodion (℥j to ℥j). The tender part may be benefited by the use of alum or tannic acid lotions. But in all cases a sufficiently broad-toed boot, with a wide sole and a low heel, is the one requirement for permanent relief.

KERATOSIS

Keratosis, or increase of the horny layer of the epidermis, is an essential part of many of the forms of dermatitis already described, such as psoriasis, chronic eczema, and pityriasis rubra pilaris. Increase of the horny layer also results from arsenical poisoning (*see* p. 993), and thickenings of apparently spontaneous origin have also been seen occasionally on the palm of the hand (*Tylosis palmæ manus*, Hebra).

KERATOSIS PILARIS

This consists of small papules, the size of a pin's head, which occur mostly on the extensor surfaces of the limbs, and are formed by accumulations of epidermis at the mouths of the hair-follicles. The hair may pierce the centre, or more often it is coiled up in the centre and broken off. The papules are often brown or black from adherent dirt.

The **Treatment** is similar to that of ichthyosis.

KERATOSIS FOLLICULARIS

This rare condition, often described as *Darier's disease*, is also one in which papules connected with the hair-follicles are capped by horny masses; and these horny masses descend as plugs into the entrance of the hair-follicle. The lesions occur especially in the groin and hypogastric region, but also on the scalp, face, and other hairy parts. The disease has so far proved incurable.

PITYRIASIS RUBRA PILARIS

This begins in different parts of the body by the formation of acuminate red papules, each with a broken hair in its centre, and surrounded by a horny collar, which dips into the follicle. They are seen best on the most hairy parts, as the arms and dorsal parts of the fingers. As they increase in number, large tracts of skin are covered by the epidermic thickening, the skin of the palms, soles, fingers, and toes is deeply fissured, and the nails are rough, thickened and broken, or thin and brittle.

Treatment.—Local sedatives may be employed, with baths and friction. Arsenic and thyroid extract have been given internally.

CORNU CUTANEUM

Horny growths, sometimes several inches in length, and generally twisted or bent, have in rare cases been seen. They are, as a rule, solitary. They consist of accumulated epidermic layers on a base of hypertrophied papillæ. The **Treatment** consists of removal and cauterisation of the base.

ICHTHYOSIS

In this disease the skin is dry and rough from thickening of the epidermis. It is congenital, though it is not seen until some weeks or months after birth, and it occasionally runs in families. In its mildest form (*Xerodermia*, dry skin) the skin is rough, dry, and dirty-looking, especially over the extensor surfaces of the legs and arms. In more pronounced forms (*Ichthyosis simplex*) the whole of the body is more or less affected, the limbs most, the scalp, face, palms, soles, genital organs, and flexures of the joints least. The skin is not reddened; but it is covered with thin epidermic scales, whose shape is more or less determined by the folds of the skin, and on the extremities inclines to a polygonal or diamond shape; and along these lines the scale is partly detached, while within them it is adherent. Still, a certain amount of shedding is constantly taking place. There is an absence of perspiration, but the sebaceous secretion is mixed with the epidermic scales, and with

the adherent dirt contributes to give a gray or greenish dirty appearance to the whole of the skin, or its most affected parts. The health is not injured by it, but the growth of the patient is sometimes stunted; and eczema is a common occurrence.

There is a gradation between this and *Ichthyosis hystrix* (porcupine skin); but the condition known under this name is often localised, or unilateral, and in some cases follows the track of cutaneous nerves. It consists of thick green or greenish-black plates or masses of hypertrophied epidermis, of square or polygonal shape, rising a quarter or a third of an inch above the skin, closely fitting together, like a mosaic pattern. Under the epidermic masses the papillæ are hypertrophied. Microscopic examination shows that in all forms the accumulated masses consist of aggregated epidermic scales. In ichthyosis hystrix the papillæ are hypertrophied, and the horny layers of the epidermis dip down into the inter-papillary spaces. In ichthyosis simplex the cutis is unaffected.

Treatment.—Complete cure cannot be effected, but considerable relief can be obtained. The scales should first be removed by hot baths, alkaline baths, friction with soap, &c., and then some emollient application should be kept constantly applied, such as glycerine of starch, vaseline, lanolin, cold cream, olive oil, &c. On the cessation of the treatment the former condition will return. For the smaller growths of ichthyosis hystrix, Crocker recommended removing the horny caps, and painting the base with a saturated solution of salicylic acid in alcohol.

WART

(*Verruca*)

Warts are small excrescences from the skin, consisting of hypertrophied papillæ capped with horny epidermis. They may be flat (*V. plana*), hemispherical, pointed, or filiform; and the larger may be lobulated, or digitate. They are generally pale pink, or yellowish, or pale brown in colour. They occur especially on the backs of the hands, and are commonest in children and young people. They often disappear spontaneously after a long time. Large warts are often seen in great numbers on the back, arms, abdomen, and neck of persons in middle or advanced life. They are greasy on the surface, and accumulate dirt which gives them a brown or even black colour (*V. seborrhoica*).

Verruca acuminata (*Condyloma*) occur on the perinæum, on the glans penis, or labia, about the anus, mouth, and other moist situations. Condylomata are generally pink or red, pointed or club-shaped, or variously modified in shape by mutual pressure, and in moist situations secrete a whitish puriform fluid. They occur as the result of irritating discharges, like those of gonorrhœa or soft sores, or as the result of friction.

Treatment.—Warts are commonly treated by the application of

nitrate of silver, glacial acetic acid, saturated solution of chromic acid, or other caustic. Saturated solution of salicylic acid in alcohol frequently applied is also effectual. Radium, Röntgen rays, and solid carbon dioxide may also be used. Thorough cleanliness and astringent lotions may suffice for the acuminate forms. Continued purgation by sulphate of magnesium (2 or 3 grains for children, or 30 grains for adults, three times a day), or by other drugs, will cause their disappearance.

Verruca necrogenica is a tubercular lesion (*see* p. 1055).

SCLERODERMIA

In this complaint the skin becomes hard, tense, and ultimately atrophic.

Ætiology.—Little or nothing is known of its causes. Localised forms have occasionally followed injury, and more general conditions have been attributed to chills, worry, specific fevers, Graves' disease, &c.

Symptoms.—Sclerodermia may be diffused or circumscribed.

In *diffused scleroderma* there is a general hardening or induration of the skin, which begins most commonly about the face, neck, shoulders, chest, and arms, and may gradually extend to the lower part of the body. There is at first no change in colour, but the skin is hard, rigid, inelastic, and cannot be pinched up into folds. As it goes on, the movements of the limbs are hindered, the joints are more or less fixed, the chest is limited in its respiratory movements, and if the face is affected it loses its power of expression; the mouth can be opened with difficulty, but the eyelids often retain their mobility. Subsequently the skin becomes shiny and glossy, irregular patches of pigment appear, and here and there are areas of vascular dilatation giving a pink or violet colour. The secretions of sweat and sebum are diminished. The course of the disease is slow, and it extends over years, eventually, in many cases, subsiding entirely. During this time the patient's health is practically unaffected, but rheumatism and cardiac troubles have been noted as occasional complications. In the skin itself eczema, erythema, and ulceration may occur.

In some cases, the disease begins with more thickening or œdema of the skin, and this, according to Crocker, tends to result in an atrophied, rigid, tight condition, which is much less liable to spontaneous recovery than the simply indurated forms.

The disease occurs in young adults and middle-aged persons, less frequently in children, and hitherto not under the age of thirteen months (*see* Sclerema Neonatorum). It is more frequent in women than in men, but little is known of its causes.

A similar condition may begin in the fingers, forming *sclerodactylia* or *acro-scleroderma* (*see* p. 662).

In *circumscribed scleroderma*, or *morphœa*, there is an unsym-

metrical patch of two, three, or more inches in diameter, frequently corresponding to the distribution of a nerve. For instance, a patch may occur over the distribution of the supraorbital nerve on the forehead; the trunk near the breast, and the limbs are also common places for the eruption. The patches are irregular in shape, or may be in the form of bands, round or along a limb. They are of a dead-white ivory colour, surrounded by a violet or pink zone of dilated vessels. The skin is smooth and dry, and may often be pinched up; it may be level with the healthy skin, or below or above it. The disease lasts several years, and then subsides and disappears, or it may extend into the diffused form, or persist in an atrophic condition. Circumscribed sclerodermia is also more common in women than in men, and can sometimes be referred to local irritation as a cause.

Anatomy.—The epidermis is unaffected except for some pigment in the rete; there is a considerable overgrowth of connective tissue in the corium and subcutaneous tissues; the deeper vessels are surrounded by numerous leucocytes; and the superficial vessels are often contracted and empty. Leucocytes also surround and may obstruct the sweat-gland ducts, and the muscular fibres of the skin are hypertrophied.

Treatment.—Very little can really be done in this disease. The patient should be kept warm at all times, and tonic remedies should be given. Locally, emollient applications and friction, and shampooing to restore the circulation in the skin, and galvanism may be employed. Thiosinamin may also be used as it is in cheloid.

SCLEREMA NEONATORUM

This is a peculiar induration of the skin, which is either congenital or begins shortly after birth in feeble infants with deficient circulation. It may begin in the lower extremities and spread to the rest of the body, or it occurs in scattered patches on the thighs, buttocks, trunk, arms, and cheeks. The affected parts feel quite hard and firm, suggesting that the subcutaneous tissue has been frozen. The patches have a well-defined edge, are slightly raised above the surface, and sometimes have a bluish-red colour. They only pit after very prolonged pressure. The children are cold and drowsy, with small pulse and feeble respiration. They often die from collapse or diarrhœa, but occasionally recover. The cause of the change is not well understood. It may be confounded with a true *œdema*, which pits readily on pressure, and in which the skin is blue and mottled.

Treatment.—The child should be kept warm and efficiently fed, by a nasal tube, if necessary.

ATROPHIC CONDITIONS OF THE SKIN

Besides senile atrophy, in which the skin becomes dry, inelastic, wrinkled, and often pigmented, the following conditions may be described as atrophy of the skin.

ATROPHODERMIA NEURITICA

This, the " glossy skin " of Paget, follows upon neuritis and other lesions of the nervous system. It is especially well seen in the fingers, of which the skin becomes smooth, shining, dry, the colour pink or red, the whole finger tapering, and the nails curved longitudinally and transversely. With this is a severe and persistent burning pain.

STRIÆ ATROPHICÆ

These are the translucent, scar-like lines which form in parts of the body which have undergone considerable distension, such as the abdomen after pregnancy (*lineæ gravidarum, lineæ albicantes*), the breasts after lactation, the abdomen, thighs, legs, and arms after extreme anasarca, and the shoulders, breasts, and thighs from obesity or the presence of more localised fatty tumours. A similar change may occur in the skin without any preceding distension, coming on spontaneously and at first without the knowledge of the patient; this may occur during some prolonged and prostrating illness, such as typhoid fever. It is then seen mostly about the buttocks, thighs, knees (*lineæ patellares*), and ankles, and may be in the form of lines or spots. In all these cases the skin is really atrophied and the elastic tissue disappears; the epidermis is thinned, the papillæ are small or absent, and the subcutaneous tissue and glands are atrophied. But an early vascular, or even hypertrophied, condition has been observed in idiopathic cases.

XERODERMIA PIGMENTOSA

(*Kaposi's Disease*)

This is a remarkable and rare disease, which consists of combined atrophy of the skin, increased pigmentation, and dilatation of the vessels. It occurs equally in males and females, and has a tendency to affect members of the same family, without being actually hereditary. It begins in childhood, with pigment spots, or with erythematous spots, which soon fade into pigment. These form over the face, neck, scalp in the temporal region, outer side

of the arm and forearm, and back of the hand. The pigment spots afterwards become atrophic, and patches of white, depressed, shrunken skin form among them. These white spots are slightly contracted, and difficult to pinch up; and subsequently sufficient tightening of the skin may occur to depress the eyelids, and set up conjunctivitis. On the atrophic area there occur pink spots of dilated vessels, which gradually enlarge. The disease may remain stationary for a long time, and may never spread to other parts of the body; but eventually warty growths develop out of either the dilated vessels or the pigment spots, and these subsequently grow into tumours of an epitheliomatous nature. These fungate, discharge or bleed; and other tumours forming in remote parts of the body, the patient is carried off by exhaustion. No treatment is of any avail.

Kaposi describes a *Xeroderma albidum* (atrophodermia albida, Crocker) affecting the leg from the thigh downwards, and sometimes the arm down to the hand, in which the skin is atrophied, and then stretched. It begins in early childhood and remains stationary.

ALTERATIONS OF PIGMENT

Increase of pigmentation is a frequent result of intense or persistent hyperæmia, through which, no doubt, there is extravasation of hæmoglobin, but the links between this and the increase of the pigment naturally in the deepest layers of the epidermis are still obscure. The most familiar instance is exposure to the sun or to the wind; but in the foregoing sections it will have been noticed how frequently pigmentation is said to follow upon the different forms of dermatitis—for instance, eczema, erythema, pemphigus, lichen, and psoriasis; to these may be added erysipelas, syphilitic eruptions and ulcerations, and especially old-standing ulcers from varicose veins in the lower extremities. The application of blisters and mustard plasters is often also followed by staining, a fact which should make one careful how one orders these counter-irritants to the neck or arms of ladies. Another common traumatic cause of increased pigmentation is the scratching which is indulged in to relieve pruritus, especially that of severe prurigo, whether idiopathic or from the presence of pediculi.

Some disorders of the skin in which hyperæmia is not a marked feature are also accompanied with pigmentation, such as sclerodermia, Kaposi's xerodermia, and leucodermia, which will be described presently. As a result of internal disease, we see pigmentation of an extreme form in Addison's disease, to a less extent in some cases of lymphadenoma, in the cancerous cachexia, in malaria, in Graves' disease, in osteo-arthritis, and in some cases

of tuberculosis, of diabetes, and of cirrhosis of the liver. Interference with the solar plexus has been suggested for its origin in lymphadenoma and in Addison's disease, but toxic causes are highly probable in many of the above and in some other disorders, such as chloasma uterinum; and pigmentation is well known as a result of the internal use of arsenic. It is not common to employ any special name, but the terms *melanodermia, melasma* (melasma suprarenale) and *chloasma* (from χλοάζω, to be pale green) have been used in different instances. In all the cases which are due to a removable cause, the pigmentation will, in its absence, eventually disappear; on the other hand, it persists in incurable cases like Addison's disease, and increased pigmentation coming on in old age does not, of course, undergo any improvement. Local collections of pigment occur, as pigment moles and pigmented warts. The special forms to be here described are lentigo or ephelis, chloasma uterinum, and ochronosis.

Deficiencies of pigmentation are seen in albinism and leucodermia.

LENTIGO

(*Ephelis. Freckles*)

Yellow, orange, or yellowish-brown maculæ appear on the face, neck, forearms, and backs of the hands, from exposure to the sun under certain conditions. They are most marked during the summer-time, and fade or entirely disappear during the winter; they are first seen about the age of late childhood, and rarely in advanced life, and they affect especially people with fair hair and blue eyes (xanthochroic type). Of a similar kind are the mottled patches of brownish-yellow pigmentation which are seen on the fronts of the legs and the backs of the forearms of those who are in the habit of sitting close to a large fire—*ephelis ab igne.*

Treatment.—As a rule freckles are better left alone: they can only be removed with the epidermis in which the pigment lies. If urgently desired this may be attempted by the method mentioned under chloasma uterinum.

CHLOASMA UTERINUM

The pregnant state, as is well known, is commonly accompanied by an increased pigmentation of the nipples, axillæ, and the line between the umbilicus and the pubes. In some women, under these circumstances, a curious band of pigmentation forms on each side of the forehead, just below, but not touching, the margin of the scalp. It is narrow in the middle line, widens out as it reaches the temple, and may extend over the zygoma on to the cheek; it is continuous, or broken into separate small patches. The colour is yellow or brown. With it may be associated the familiar dark ring

round the eyes. This frontal chloasma sometimes recurs with each successive pregnancy, and disappears with delivery. It may be due to other uterine disturbances—*e.g.*, dysmenorrhœa—and sometimes no cause can be traced.

Treatment.—Mercury perchloride has been most used locally, in a solution of the strength of one or two grains to an ounce of almond emulsion, applied twice daily until the skin is reddened. Zinc ointment may then be applied. Solutions of citric acid, carbolic acid, and other mild caustics, by which the epidermis is removed, and with it the pigment, have also been used. But it tends to recur. Crocker recommended salicylic acid paste, or plaster, or a saturated solution of the acid in alcohol kept on for some hours.

OCHRONOSIS

In this rare condition, called by Virchow *Ochronosis*, there is a black pigmentation of the skin, cartilages, and sclerotics; but whereas the cartilages have been constantly stained, the skin has been affected in only a few instances. The face is of coal-black or dark brown colour, darker than that of Addison's disease: the hands may present bluish-black areas, and patches have been seen on the mucous membrane of the lips. A black patch is seen in the sclerotics on each side of the cornea, midway between it and the canthus. The change in the cartilages is clinically observable in the ears, which have a bluish-gray colour due to the blackened cartilage being seen through the thin skin; but post mortem the rib-cartilages, and the intervertebral, sterno-clavicular, laryngeal, and tracheal cartilages have been found of jet-black or inky-black colour.

Some tendons have been smoky brown in colour, and the cardiac valves and chordæ tendineæ discoloured in patches. The pigment is deposited in the matrix of the cartilages and in the fibrous tissue of the corium of the skin.

Some of the cases have been associated with *alkaptonuria* (*see* p. 895); and a few with carboluria after the constant application of carbolic acid to chronic ulcers for many years.

ALBINISM

This is a congenital deficiency of colour, not only in the skin, but also in the hair and in the iris and choroid. It is at once recognised by the white hair and the pink eyes; and there is commonly intolerance of light (*photophobia*), and it may be nystagmus, from the want of pigment in the fundus of the eye. It occurs in dark races as well as in the pale-faced; and in various animals—cats, mice, and others.

LEUCODERMIA

Scattered white patches, occurring in any part of the body—the neck, chest, abdomen, arms, or legs—are known as leucodermia. The patches vary from half an inch to several inches in diameter, are irregular in shape, but have convex borders, are frequently grouped together, and, gradually enlarging into one another, may form convex scalloped borders. Around the margin of each patch the skin is sometimes, but not always, darker than normal, and the colour gradually fades as it is farther and farther from the white patch, until the normal tint is reached, some half-inch or so away from it. There is thus in leucodermia a double process : a deficiency of pigment over a certain area, and an increase of pigment around it. Beyond these alterations of the pigment, the skin is quite unchanged ; but hair growing from leucodermic patches may also lose its colour. The disease is an acquired one ; it is more common in hot climates, and among dark races, than in England. It has been seen rather frequently in association with morphœa, alopecia areata, Addison's disease, and exophthalmic goître ; and by some it is regarded as having a definitely neurotic origin.

Diagnosis.—If it is seen on the neck it may have to be distinguished from the rare pigmentary syphilide, which occurs on the back of the neck in women, and has a dappled appearance of sharply cut oval white spots close set on a dark brown ground. Otherwise the perfectly smooth skin and the bright white areas with convex margins advancing towards the pigmented skin are distinctive.

Treatment.—Improvement may take place spontaneously ; but treatment is of little avail. Unsuccessful attempts have been made to develop pigment on the white patches by blisters, ammonia, or other irritants. Staining with walnut-juice may be used to mask it temporarily. On the other hand, the pigmented parts may be treated in the same way as chloasma.

DISEASES OF THE SWEAT-GLANDS

ANOMALIES OF SECRETION

Anidrosis, or deficiency of perspiration, occurs in fevers, in diabetes, and in some diseases of the skin—*e.g.*, ichthyosis and pityriasis rubra.

Hyperidrosis, or excess of perspiration, may be general or local. General sweating occurs as a result of dilatation of cutaneous blood-vessels, as after exercise, or from emotional causes. Sometimes, on the other hand, it occurs with contracted vessels, as in conditions of collapse or fear. General perspiration has been mentioned in

connection with malaria, phthisis, pyæmia, and the crisis of acute illnesses. Local excess of sweating occurs from emotional causes and in rickets. A very troublesome form of excessive sweating occurs about the hands and feet, axillæ, and genitals in some persons without any adequate cause. Some such sufferers are in deficient general health, but others are perfectly well.

The local application of belladonna liniment or the use of atropine or belladonna internally should be tried. A drop dose of liquor atropinæ will sometimes stop the sweating of phthisis for two or three successive nights. Local sweating may be also treated in the same way, or by some of the methods used for the next complaint.

Bromidrosis.—This is often associated with hyperidrosis—that is, the sweat is both offensive and excessive. It affects chiefly the feet and axillæ; and the odour is probably due to the decomposition of the fatty sebaceous material which is secreted with the sweat. It is not uncommon in young men or young women of the domestic class; and it may be quite independent of the general health. Thin has described a bacterium in connection with it—*bacterium fœtidum*. It is essential to wash the feet thoroughly and frequently, and use astringents and antiseptics. The socks may be dusted inside with finely-powdered boric acid, and should be frequently changed; or with a mixture of salicylic acid, 3 parts, starch powder 10, and talc, 87 parts; or the feet may be painted with a 5 per cent. solution of chromic acid; or smeared with a salicylic ointment, of 2 per cent. strength, or an ichthyol ointment of 5 per cent. strength.

Chromidrosis, or coloured sweat, is a rare affection, and is perhaps sometimes due to indican. But the possibility of its being feigned should always be remembered.

Hæmatidrosis, or sweating of blood, also quite rarely occurs, mostly in highly neurotic people.

Uridrosis is the name given to some cases in which the sweat has crystallised on the surface, and the crystals have been found to contain urea and salts. It has been seen in Bright's disease (*see* p. 912) and in the stage of collapse of cholera.

MILIARIA

This name is given to rashes determined by profuse secretion of sweat, such that it is unable to escape by the ducts, and either raises small vesicles in the epidermis or sets up a local inflammation.

Miliaria crystallina or *sudamina*.—In this form there are small transparent vesicles, not larger than a pin's head, due to the elevation of the most superficial layer of the epidermis by accumulated sweat. They are found most abundantly on the chest and abdomen, but from their perfect transparency may be better felt than seen. The vesicles dry up, and leave a few branny scales, the remains of the detached epidermis. They are most common in phthisis and in enteric fever.

In *miliaria rubra*, there are vesicles produced in the same way, but accompanied by inflammation. They are surrounded by a red areola, and contain a yellow turbid alkaline fluid or actual pus. It is not uncommon to see these in the course of rheumatic fever.

Sweating in infants, as a result of wrapping them too closely in binders or napkins, produces a papular form of miliaria (formerly called strophulus or red gum); and the prickly heat of hot countries (formerly *lichen tropicus*) is regarded as *miliaria papulosa*. It affects chiefly the trunk and thighs, is accompanied by severe itching, and does not materially influence the general health.

DISEASES OF THE SEBACEOUS GLANDS

SEBORRHŒA

Under this term, meaning excessive secretion of sebum, many affections have been described. Two of them are the forms of pityriasis above mentioned (*see* pp. 1050, 1051). Another is the *vernix caseosa*, a waxy covering of the fœtus, which is often left adherent to the scalp for some time after birth, and consists of epidermic scales accumulated during intra-uterine life.

In *seborrhœa oleosa* the face appears constantly greasy, or moist, and consequently shiny; if the finger touches it, it is obviously moistened by the greasy secretion; the face, moreover, gets readily dirty from the adhesion of the particles of dust floating in the air. It is most frequent about the forehead and nose, but occurs on all parts of the face. It varies from time to time, being, perhaps, aggravated by gastric troubles, and by general ill-health. It is often associated with the above forms of pityriasis and with acne.

Treatment.—The general health should be attended to; and the face should be frequently washed. In cases accompanied by acne, mild sulphur preparations may be used.

COMEDO. ACNE

Not uncommonly the sebaceous follicles get blocked by their secretion, and thus lead to a prominent papule on the skin (*comedo*), or to inflammation around the distended follicle (*acne*).

The comedo is commonly seen as a whitish conical swelling on the forehead, cheeks, or nose, with a minute black spot on the summit. The swelling is due to the accumulated and inspissated sebum; the black spot is adherent dirt. It is frequently accompanied by seborrhœa. If pressure be made upon the base of the papule with a finger-nail on either side, a plug of sebum is extruded, and occasionally in this sebum can be found an acarus, the *Demodex folliculorum;* but much more constantly the *Bacillus acnes*, a Gram-

positive organism growing on anaerobic media, is seen, and sometimes micrococci.

Comedones may persist sometimes without much change, fresh follicles being involved from time to time; but generally some of them become inflamed so as to constitute acne, and often the great majority of the lesions are quite early of this nature.

In *acne* the papule tends to be larger, is conical in shape, pink or red in colour, and as suppuration takes place within the follicle, a pustule forms at the summit. Eventually the pustule bursts, and the redness subsides, leaving no trace; but the lesion is sometimes so extensive, and the suppuration so deep, that a well-marked scar is left. This is especially likely to be the case where the papule is irritated by the friction of the clothes, as, for instance, on the back and shoulders, when pyogenic micrococci may also have a share in the lesion.

The ordinary form is called *Acne vulgaris;* large hard inflamed papules constitute *A. indurata;* those which have suppurated freely, *A. pustulosa.* Comedo was formerly called *A. punctata.*

Acne vulgaris affects especially the face, chest and shoulders, the back of the chest and shoulders often presenting the largest pustules and the most numerous and extensive scars, while the front of the chest is the least involved.

It is a disease of early adult age, or puberty; beginning in young men before the beard has begun to grow, and commonly subsiding after it has grown, and rarely lasting after the age of thirty. It also occurs in young women, but less commonly and less severely than in men. Beyond these relations of age there is not much positively known as to the cause of acne. Exposure to dirt and grease, as amongst some classes of artisans, especially those having to do with tar, no doubt produces it by direct obstruction of the sebaceous follicles; but where that cannot be alleged as the cause, its origin is mostly inexplicable. Staphylococci are found when suppuration has occurred, but that they are the originators of the condition is not clear. A certain amount of anæmia is not infrequently present.

Acne varioloformis is a rare pustular eruption which occupies the forehead, scalp, and temples, and leaves scars deeply pitted like those of small-pox. According to Sabouraud it is due to staphylococcus aureus.

Treatment.—Comedo should be treated by thorough washing with soap and water night and morning, and drying with a rough towel, by which means already some of the plugs will be removed. Others may be pressed out by placing over the top of each papule a watch-key, or other key, and pressing firmly until the plug is extruded. Special instruments have been devised for this purpose. Both in this and in acne any condition of ill-health may be dealt with, and it is common to give mild laxatives and iron, as they are believed to be of value even when no anæmia or digestive fault is in question. The sulphates of iron and magnesia in peppermint-water form a very

good mixture for this purpose. Other tonics may be suitable in special cases, and the diet may as well be regulated. Where there is definite acne, the washing and friction may still be carried out; pustules should be opened with a needle or lancet, and some stimulant preparation should be applied. This may be left on only during the night, as the course of the disease is always slow, and the inconvenience of having ointments on the face during the day may be respected. Sulphur is one of the most useful of local remedies; it may be used as iodide of sulphur ointment, which is effectual, but temporarily stains the skin; sulphur suspended in liquor calcis: ointment of precipitated sulphur (2 or 3 drachms in an ounce of vaseline); or a lotion of sulphide of potassium (1 to 40). If there are many pustules, mercurial ointments are also valuable, such as white precipitate, or the acid nitrate; but with much dermatitis between the acne pustules, soothing remedies, like zinc and lead lotions, may have to be temporarily employed. In any case, the treatment must be perseveringly continued.

Acne has been recently treated with much success by the injection of vaccines of sterilised cultures of the acne bacillus and of staphylococci, in association with observations on the opsonic index (*see* pp. 20, 539). Vaccines should, if possible, be made from the patient's own organisms. Fresh brewer's yeast may be given also, a tablespoonful twice a day, to increase the opsonic index.

BOILS

(*Furuncles. Furunculosis*)

These are local infections by staphylococcus pyogenes aureus, starting in the neighbourhood of the hair-follicles. They are common on the back of the neck, the trunk, and the buttocks; and they may be caused by the pressure, friction, and irritation of clothes. There is often an antecedent state of ill-health, such as that present in convalescence from acute illnesses, and in diabetes, when the resistance to organisms is less than normal. The opsonic index shows that there is a diminished resistance to staphylococci.

Symptoms.—The trouble begins with a painful red and slightly raised spot or pimple; which gradually enlarges, and becomes hard, infiltrating the surrounding skin, and extending deeply at the same time. The colour becomes dusky red, and ultimately the apex softens and a little pus is extruded, and later a whitish-yellow slough of necrosed tissue. When this has been discharged, the inflammation subsides, and the sore gradually heals by granulation, leaving a small scar.

In some persons, boils are constantly forming in all parts of the body, and this may go on for months or years (furunculosis).

Treatment.—Glycosuria or other general predisposing cause should, of course, be treated: otherwise, tonics such as iron, arsenic,

and quinine may be given. Locally, when pointing the boil should be incised, and dressed with boric lint wrung out of hot water.

For the repeated occurrence of boils, the treatment by vaccines of staphylococci has been often most effectual. The vaccine is best made from the organism found in the boils of the patient under treatment: and from 75 to 200 millions of organisms may be injected every ten days.

CARBUNCLE

This is a similar infection by staphylococci, leading to extensive inflammation, induration, and necrosis of the skin or subcutaneous tissue. It differs from a boil in its extent, and in the fact that there are numerous foci of suppuration, leading to discharge through several openings instead of one. The patients are generally of middle age or older; and diabetes mellitus is a very common antecedent. The common seat of the lesion is the back of the neck, but it also occurs often on the back. It begins as a flat indurated area, of red or purplish-red colour; this extends, and becomes more elevated above the level of the skin, spots of softening occur, and discharge of pus takes place. The hard mass forms a slough, which comes away, leaving a deep ulcer; and this ultimately heals by granulation, leaving a scar. A carbuncle may be from one to three inches in diameter, and occasionally very much more. There is much constitutional disturbance, with pyrexia: septic infection is very likely to occur, or death may take place from exhaustion.

Treatment.—This is largely surgical. If recognised early carbuncle may be injected with carbolic acid: at a later stage it has been customary to relieve tension and facilitate separation by making a crucial incision, and dressing with boric acid fomentations; but entire removal by the knife, or scraping out and applying pure carbolic acid, may be practised.

MILIUM

This is a small bright white round tumour, the size of a pin's head or slightly larger, which results from complete obstruction of the duct of a sebaceous gland. Such little tumours occur on the forehead, eyelids, cheeks, and genitals. Occurring in children they were formerly called *strophulus albidus*. Sometimes they occur in great numbers on thin cicatrices, especially those of lupus. The contents are cholesterin and fatty material. They can be treated by puncturing the skin and squeezing out the contents.

ADENOMA SEBACEUM

This forms small tumours from the size of a pin's head to that of a pea, situated on the middle portion of the face, round, firm, solid and white, yellowish white or brown. If pricked, white inspissated

sebum can be squeezed out. In addition there is often some telangeiectasis over and around them. The condition is one of hypertrophy of sebaceous gland tissue, and is probably congenital in its early stages. It must be treated by electrolysis or excision.

SEBACEOUS CYSTS

(*Wens*)

These are retention cysts arising from obstruction to the ducts of the sebaceous follicles. They are most common on the scalp, but occur on the eyebrows, face, or neck, and more rarely on the trunk or limbs. In size they vary from that of a pea to that of a nut or an orange. They are hemispherical, or more globular, uniform and smooth. The skin looks thin, and often presents well-marked vessels ramifying over it. The contents are semi-fluid, or pasty, and consist of animal fats, albumin, epidermic cells, cholesterin, and earthy salts, enclosed in a capsule made up of layers of epithelial cells and fibrous tissue. The **Treatment** consists in incising the cyst, squeezing out the contents, and tearing out, or dissecting out, the cyst wall entirely.

DISEASES OF THE HAIR AND HAIR-FOLLICLES

The hair may be developed to an excessive degree as a congenital phenomenon; this is very rare, and is called *hirsuties*. Deficiency or falling of the hair, known as *alopecia*, is, on the other hand, exceedingly common. The following three conditions are quite rare: *Trichorrexis nodosa*, in which some of the hairs are found to present little nodules or thickenings due to the splitting up of the cortical fibres; *monilethrix*, in which the hair looks beaded. and breaks readily at the internodes, so that it is only two or three inches long all over the head; and *lepothrix*, in which the hairs are brittle, and present irregular masses on and around them.

The most common *change of colour* in the hair is that known as *canities*—the hair gets successively grey and white. This is a senile change which may occur prematurely. But occasionally sudden whitening of the hair occurs after fright, intense emotion, or in consequence of neuralgia. It is probably due to the development of air-bubbles, which conceal the pigment. The colour sometimes returns spontaneously, but nothing can be done for it.

Of the above ailments, *alopecia* will be more fully described, and afterwards *sycosis*, or inflammation of the hair-follicles. The hair is also involved in some of the parastic diseases of the skin.

ALOPECIA

Baldness is most familiar to us as it appears in old age (*senile alopecia*), though it occurs also in quite young people (*premature alopecia*). Different degrees of atrophy of the hair-follicles and structures of the skin have been found in old cases of baldness, but its immediate antecedent is doubtful. Premature baldness is not explained by mental exertion, wearing tight hats, insufficient lubrication of the scalp, &c., but it is certainly sometimes hereditary, and it is constantly associated with the presence of pityriasis capitis or seborrhoic eczema, so that micro-organisms are now often regarded as the primary cause. The hair thins first at the back of the vertex, and at the front part of the temples. Ultimately there is only a fringe left round the temples and occiput. A temporary alopecia is caused by transient interference with the nutrition of the hair-bulbs; thus the hair falls not infrequently after fevers, after erysipelas of the scalp, in the second stage of syphilis, and in the parasitic diseases which will presently be described. A localised alopecia is a necessary part of any scar following ulceration deep enough to destroy the hair-follicles.

Premature loss of hair is practically incurable, though it may be checked or prevented by adequate treatment of the pityriasis or other disorder of the scalp which precedes it. Restoration of the hair after fevers, &c., may be hastened by the use of tonics internally, faradising by means of the wire brush, and by the use of stimulants, such as oil of mace, cantharides, and ammonia, which will be again mentioned under the next complaint.

ALOPECIA AREATA

(*Area Celsi*)

This peculiar form of baldness occurs at almost any age, but is commoner in children and young people. The sexes are about equally affected, and in the majority of cases there is no indication of ill-health. Quite unexpectedly it may be noticed that there is a bald spot on the head, the size of a sixpence or larger, and this gradually extends. The spot is circular, and, as nearly as possible, perfectly bald, smooth, and shining. But one feature is very constant—namely, that just at the edge of the patch, and less commonly in its middle, are observed a few—from one to ten, or more—short, dark stumps, from $\frac{1}{8}$ to $\frac{1}{4}$ inch long, and thicker at the free end than at the root. If extracted and placed under a microscope, the free end is seen to be brush-like and broken up into its constituent parts; while there is no bulb, but only an atrophied root. No indications of a specific fungus can be found. Other bald patches may form on the scalp adjacent to the first, or elsewhere, and two or more may coalesce to form an irregular figure. The patches persist some months or a

year, and gradually become covered with hair, so that complete recovery takes place.

The **Pathology** is still obscure. Histological examination of the skin has shown atrophy of the hair-follicles, and round-cell infiltration of the outer root-sheath, the surrounding skin, and subcutaneous tissue. The views held with regard to it are—(1) that it is a parasitic disorder, chiefly on the evidence of alleged cases of contagion, and of its occasional association with ordinary ringworm; (2) that it is a disorder of nervous influence—atrophoneurosis; (3) that it is the result of seborrhœa (Sabouraud), which may itself be due to micro-organisms.

The **Diagnosis** presents no difficulties, as ringworm is rarely so completely bald, and the stumps of hairs have a different appearance.

Treatment.—This consists in such treatment of the general health by tonics as may seem indicated, and the application of local stimulants and irritants—such as ammonia liniment; or tr. cantharidis in water (1 to 8); or acetum cantharidis; or tr. cantharidis ʒss, carbonate of ammonium gr. xl, and sp. rosmarini ʒss, with water to 8 ounces; or ol. myrist. express. ʒij, ol. olivæ ʒvj. One of these should be rubbed in every night, to the extent of producing slight redness only. The faradic brush, occasional blisters, strong liquor iodi, or high-frequency currents, may also be employed.

UNIVERSAL ALOPECIA AND CONGENITAL ALOPECIA

In the former the hair falls from every part of the body, scalp, eyebrows, axillæ, and pubes, so that not a hair is left. As a rule, no cause can be assigned, and the cases are incurable.

In the latter, the nails are wanting as well as the hair. These cases may be hereditary.

SYCOSIS

(*Coccogenic Sycosis. Folliculitis*)

This is a chronic inflammation of the skin of the beard and hairy parts of the face, beginning in or around the hair-follicles, and due to infection by micro-organisms, especially the staphylococcus aureus and albus. The infiltration is deep-seated, pus forms in the follicle, and the hair is loosened. If the pus escapes it dries up into an adherent crust. The pustules may be at first isolated, but the intermediate skin is involved, and considerable infiltration takes place, the part being red, irregular, and nodulated, with pustules and adherent crusts. The disease occurs exclusively in adult males; can be transmitted from one person to another, as, *e.g.*, by the shaving-brush; and is exceedingly obstinate.

A somewhat similar but rarer condition arises from the action of the ringworm fungus (*see* p. 1081). In this the extraction of the hairs is less painful, and the fungus can be readily recognised under

the microscope. Sycosis may be also confounded with eczema, but in this there is more itching, the lesions are more superficial, and generally extend beyond the limits of the hairy parts.

Treatment.—The hair must be cut close, and the crusts must be detached by softening with poultices or oiled lint.

The hairs which are loosened by inflammation must be pulled out, and subsequently it may be necessary to extend the epilation to others. The inflammation may be allayed by lead, zinc, or other mild antiparasitic ointments, applied thickly on linen, or by ammoniated mercury ointment. As the hair grows it must be constantly cut, and epilation steadily continued. When the infiltration has subsided, and the pustules are less numerous, the hair may be shaved. The Röntgen rays may be used for the removal of the hairs, as in ringworm.

Antistaphylococcal vaccines are also employed, as in acne and boils.

VEGETABLE PARASITES

The following diseases of the skin are due to vegetable parasites belonging to the class of *Hyphomycetæ*—*Tinea versicolor*, *Erythrasma*, *Ringworm*, and *Favus*. The parasites, or fungi, consist of jointed rods or threads, the mycelium; and round or oval bodies, the conidia, or spores. Their average size is greater than that of the bacteria, and they can generally be well seen with a moderately high power of the microscope (one-fifth inch), after the hair or epidermic scales with which they are connected has been rendered translucent by liquor potassæ. Hairs may be stained in the following manner: Wash the hair in ether, then place in solution of gentian violet (5 per cent. in alcohol); steep in iodine to fix the stain, decolorise in anilin oil, then keep in pure anilin for a few seconds, wash in xylol, and mount in xylol balsam (M. Morris).

The fungi have also been cultivated on various media by bacteriological methods, and their distinctive characters thus more fully brought out.

TINEA VERSICOLOR

This is a common affection of the skin, produced by contagion, and fostered by warmth and moisture. It is more frequent in men, and especially in those who wear flannel underclothing. It is not often conveyed directly by contact—for instance, from husband to wife.

The disease begins as a small circular spot, of a yellowish-brown colour, which is slightly raised above the skin, and from which a few whitish scales can easily be detached by scraping with the finger-nail or a scalpel. The patches extend, and fresh ones form,

so that soon a large part of the chest, where it is commonly seen first, is covered with a brown, or brownish-yellow, irregular patch, with a convex or scalloped margin; and on the healthy skin adjacent are numerous small isolated patches from a quarter to half an inch in diameter. The disease occurs only on covered parts, and is most abundant on the front and back of the chest and the abdomen. The scrapings examined in liquor potassæ under the microscope show epithelial plates with the specific fungus, *Microsporon furfur*, forming a network of branching mycelium threads, scattered among which are little groups of the relatively large conidia, like bunches of grapes. The affection does not cause much trouble beyond some itching, and is frequently ignored by the patient; but the great extent which the discoloration may sometimes reach has led to its being mistaken for Addison's disease and other pigment affections. The peculiar colour, the convex edge, and the ready desquamation of the surface should be quite distinctive, and the diagnosis is at once confirmed by the microscope.

Treatment.—It is quickly cured by rubbing in lotions of sodium hyposulphite (1 in 8) or of sulphurous acid (1 in 4), or finely powdered borax. It may, however, return if the same underclothing is used without thorough washing and disinfection.

ERYTHRASMA

This disease is allied to tinea versicolor, but is much less common. It produces rough, brown scales, and occurs chiefly between the scrotum and thighs, in the axillæ, and mammary folds. It is due to a fungus, the *Microsporon minutissimum*. It is more common in men than in women; it spreads very slowly, and lasts a long time. It must be treated like tinea versicolor.

RINGWORM

The diseases which are commonly included under this term are ringworm of the head (*Tinea tonsurans*); ringworm of the body (*T. circinata*); ringworm of the beard (*T. sycosis*); and Burmese ringworm (*T. marginata*). They are dependent upon the growth of two, if not three, closely allied fungi. It will be better to describe the clinical features of the various forms first, and state what is known of the organisms afterwards.

Tinea tonsurans (*Ringworm of the head*).—This disease is the great scourge of schools, and allied institutions, among the poorer classes. It is frequent in children, rare in infancy, and not easily caught by adults. It spreads by contact, and by the use of hats, caps, brushes, combs, and towels in common.

It generally first appears as a round patch, on which the growth of hair is much thinner than elsewhere. On close examination the

skin is seen to be pink, perhaps a little swollen, and covered with minute branny scales. Besides the thinly scattered long and healthy hairs are seen a number of broken stumps of hair, opaque, black or dark brown in colour, twisted and bent. If an attempt be made to extract one of these broken hairs with a pair of forceps, it will almost certainly break off short; if then placed under the microscope, and moistened with a drop of liquor potassæ, it will be seen that the natural structure of the hair is unrecognisable. The substance is quite opaque, and the hair seems to be converted into a mass of fungus spores (conidia). This is, however, only a sheath of spores surrounding the hair, which is itself occupied chiefly by mycelium tubes running in a longitudinal direction. These may be more readily observed in hairs which are less completely diseased.

The patch spreads by the implication of hairs at its circumference, and fresh patches form in other parts of the scalp. As these enlarge they become more completely denuded of long hair, though they nearly always present a considerable quantity of the short stumps which have been described; and these may be surrounded and mixed with scabs, crusts, or sebaceous matter, or with a fine whitish powder, of which probably the fungus elements form a part. The patches spread slowly; some may heal in the centre as they extend at the edges; or the patches may coalesce, and nearly the whole scalp may be affected. Sometimes, on the other hand, one or two patches persist, without improvement, but without spreading. The disease may last for years, but eventually dies out, and the hair is perfectly restored. There is rarely much inflammation; but occasionally the hair-follicles inflame, coalesce, and form a red or pink swelling which is soft and boggy to the touch, and discharges pus from a few points; the hairs are loosened and fall out, and the patch may remain bald when the other parts of the scalp have recovered. The condition is called *kerion*.

Probably the fungus invades the hair close to the scalp, and pushes down towards the bulb. The hair in the follicle is thus weakened or destroyed, and as it is forced outwards by the newly-formed epithelial plates, it breaks off. The newly-formed epithelium is, in its turn, invaded as soon as it gets into the horny condition. It was shown by Thin and myself that the fungus only invades the hair itself, and cannot be found in the cells of the root-sheath or in the structures of the hair-follicle.

Tinea circinata (*Ringworm of the body*).—This occasionally co-exists with tinea tonsurans, but often occurs alone. The fungus invades the epithelial scales and downy hairs of the skin and produces a circular patch, from half to one inch in diameter, slightly raised above the surface, sharply defined, pink in colour, often papular, and covered with fine scales. If the surface be scraped with a scalpel, and the scrapings be placed, with a little liq. potassæ, on a glass slide, the mycelium and spores of the fungus will be seen. The patches increase by extension at the circumference, and may, as they spread, heal in the centre. Occasionally a few vesicles

form on the surface from the irritation of the parasitic growth; thus in part justifying the former name, herpes circinatus. They occur on the face, neck, and arms most frequently; they are, as a rule, few in number, and may be solitary.

Tinea marginata (*Tinea cruris, Eczema marginatum,* or *Burmese ringworm*).—This is confined to adult males, and occurs chiefly about the inner sides of the thighs, genitals, and groins of those who are constantly sitting, such as horsemen and cobblers, but it may invade the axilla. It begins in spots or rings, which, spreading at the margin and recovering at the centre, unite together, and ultimately extend on both sides symmetrically. It forms a broad yellowish or brownish-red band, which runs in a curved or gyrate form along the inner side of the thigh and scrotum to the inner side of the buttocks, and over the groin and lower part of the abdomen. The lesions are much more extensive, more inflamed, and more obstinate than those of tinea circinata.

Tinea sycosis (*Ringworm of the beard. Hyphogenic sycosis*).—The hair-follicles of the chin and cheeks are here inflamed by the presence of the fungus; they suppurate, and the hairs become loosened. Induration and swelling of the intermediate skin also occur. It differs from sycosis, already described (*see* p. 1077), in that it first attacks the hairs, and loosens them early, so that their extraction is painless; it spreads more rapidly and produces deeper infiltration. Microscopic examination shows the fungus, in which the mycelium is more abundant than in the spores.

Tinea of the nails.—The invasion of the nails by a fungus is called *onychomycosis,* and this may be a ringworm or favus, either of which is probably conveyed by scratching other affected parts. The nail tends to become elongated and curved over the end of the finger, with a thick edge, rough uneven surface, and dirty yellow colour; it is also brittle and readily splits. If fragments or scrapings are soaked with liq. potassæ, and examined under the microscope, chains of spores of the fungus are seen.

The Fungi of Ringworm.—The point upon which a separation of these fungi is based is the size of the spores. Tinea tonsurans is in England chiefly due to a variety in which the spores are small, measuring from 2μ to 4μ. It is called *Trichophyton microsporon,* or *Microsporon Audouini.* As usually seen, the shaft of the hair is penetrated by the mycelial threads and the spores form a dense sheath, five or six spores deep, around the hair; and this sheath is continued beyond the follicle on to the stump or fragment of the hair which still remains. The common ringworm, caused by the *microsporon,* is almost confined to children, is very contagious and very obstinate, so that months may elapse before it is cured; but it ultimately gets quite well.

The other ringworm fungus is named *Trichophyton megalosporon.* The spores measure from 3μ to 6μ, commonly 5μ, and are thus larger than those of microsporon; they attack the root of the hair first and grow upwards. Two varieties have been described: in one,

Trichophyton endothrix, the spores and mycelium are contained entirely within the shaft of the hair; in the other, *T. endo-ectothrix,* the spores form a sheath outside the hair (as they do in microsporon), and only the mycelium tubes lie within it. But these distinctions are not universally accepted. Cases due to megalosporon are much more amenable to treatment, and those due to the ectothrix variety more than those due to endothrix. As to the further relations between the fungi and the clinical forms of disease, very different statements are made, which are in part explained by the different prevalence of the fungi in countries remote from one another. In England the microsporon causes the large majority of cases of ringworm of the head; trichophyton megalosporon endothrix is the chief cause of tinea circinata, and of tinea of the nails (onychomycosis); and T. megalosporon endo-ectothrix occurs in tinea sycosis and in a few cases of tinea tonsurans, and is the chief cause of kerion. Though contagion is, especially in ringworm of the scalp, mostly from child to child, the microsporon and megalosporon endo-ectothrix are sometimes caught from the horse, dog, cat, cattle, or birds.

Tinea marginata is due to another parasite, epidermophyton inguinale (Sabouraud).

Treatment.—The principles of treatment in *Tinea tonsurans* are (1) destruction of the spores of the fungus by means of parasiticides locally applied; (2) the complete removal of the hair from the affected area (*epilation*), so that no further material remains as a soil for the fungus.

The difficulty of the first method lies in the fact that the spores multiply within the hair-follicle, and are, therefore, to a large extent, protected from the parasiticides employed. It may succeed in slight cases, but in the severer cases it generally needs the assistance of the second procedure; and indeed the stronger parasiticide applications themselves loosen the hairs by inflaming the follicles and the surrounding skin.

The Röntgen rays have proved to be the most efficient means for loosening the hairs, and a cure can be effected by their means in one-tenth the time that is necessary in the older methods.

In either case the first thing is to cut the hair quite close, and remove all crusts and scabs by soaking with oil and subsequent removal and washing. The extent to which hair is removed may vary with circumstances. For local applications complete shaving is best; but where it is desirable to save appearances a fringe of hair may be left all round the head, or with a single patch the hair may be close cut or shaved for an inch or more round it.

Röntgen rays.—These have to be applied with great care and precision in order to avoid injuring the skin, and at the same time to get the full depilatory effect at one sitting. For this purpose the duration and strength of the application can be regulated by *Sabouraud's pastilles,* which are discs of paper thickly coated with an emulsion of platino-cyanide of barium. If the source of the

rays is placed 15 centimetres from the scalp, and the pastille between them 8 cm. from the anode, the desired amount of influence is reached when the pastille acquires a particular fawn tint identical with a test colour supplied. The application is made to a limited area of the scalp at one time, the rest being protected by sheet lead; but time is saved by treating on the same day five points, situated at least five inches from one another. After fifteen days the hair comes out with the slightest traction; and the patch must be constantly washed with soap and water till it is all removed, the diseased hairs falling last. The head remains bald for five or six weeks, when finer and coarser hairs begin to grow.

Parasiticides.—These must be regularly and constantly applied. The patch should be washed clean with soap and water night and morning, and the ointment or paste well rubbed in.

The following are some of those most commonly employed: Mercurial applications, such as ung. hydrarg. ammon.; oleate of mercury, from 3 to 10 per cent.; glycerinum acidi carbolici, or the same with a larger amount of carbolic acid; sulphur ointment; creosote; tincture of iodine; thymol or turpentine. Some of these may be combined together: sulphur 2 and carbolic acid 1 in 16 of lard or vaseline; iodine 1 and creosote or oil of cade 3 parts; boric acid ℥j with methylated ether ℥x, oil of rosemary ℥ij, and methylated spirit to ℥xl (Aldersmith); caustic potash 9 grains, carbolic acid 24 grains, in ½ oz. each of lanolin and ol. cocoæ (Harrison); formalin in 40 per cent. solution, rubbed in vigorously with a brush ten minutes on alternate days for four days (Salter), is sometimes efficient, but it seems to be painful; sulphur ℥j, salycilic acid, β-naphthol, and ammoniated mercury, of each 10 grains, and lanolin, ℥j (Jamieson). Kerion may be treated with weak lotions of lead or boric acid, and gradually subsides; it should never be incised.

In order to test the effect of treatment the hairs must be examined from time to time, but no case can be considered cured until a good crop of hair has grown over the whole scalp, and even then a very careful search must be made for still active disease, which may be shown by a broken and twisted hair, or a small brown scaly spot.

During the treatment of a child with ringworm great care should be taken to prevent its spread to other children. The patient should use a separate brush and comb, and towels. The head should be covered with a cap, which may be freshly lined with a piece of tissue paper every day, the old piece being destroyed.

Tinea circinata is easily cured by the use of ung. hydrarg. ammon., oleate of mercury, tincture of iodine, sulphurous acid in solution (one part to two or three of water), a weak carbolic acid glycerine, hyposulphite of sodium (1 in 8), or some other not too strong parasiticide.

Tinea marginata requires the constant use of similar parasiticides. *Tinea sycosis* may be treated also with Röntgen rays or by epilation.

In *onychomycosis* the nails should be scraped thin, softened with

alkaline solutions, and soaked in lotions of sulphurous acid, sodium hyposulphite, or mercury perchloride (2 grains to 1 ounce of water); or they may be frequently painted with carbolic acid.

FAVUS

In this disease, rare in England, the fungus attacks the epidermis and the hair-follicles: it may at first form patches like those of ordinary ringworm, but soon there appears a small bright yellow circular disc, with a depressed centre and a gradually thinning margin. This, the "favus cup," is caused by the fungus elements separating the layers of the epidermis and lifting them up, except at the central point where the hair-follicle joins the skin. This characteristic lesion may occur on the scalp, or on any other part of the body, the forearm for instance, determined by contagion; and it is conveyed not only from man to man, but to man from domestic animals—rabbits, dogs, cats, and others. When numerous cups have formed they become aggregated together, and form a thick continuous yellow crust, with an irregular honeycombed surface, giving off an offensive odour resembling that of mice. The hair-sacs are destroyed, the hairs fall out, and baldness results; moreover, the favus masses often become a nidus for pediculi, and eczema and impetigo complicate the original lesion. If the masses are examined under the microscope after soaking in liquor potassæ, the mycelium and spores (conidia) of the *Achorion Schönleinii* are seen. The conidia are larger and more varied than those of the ringworm fungi, and the mycelium is shorter and more jointed.

The *nails* may be also invaded by the fungus of favus. They present an appearance similar to that in ringworm (*see* p. 1081), and sometimes a distinct cup forms under the nail. The nail may be examined in the same way.

Treatment.—The crusts must be softened by oil or poultices, and removed; parasiticides and epilation are needed here, and for most cases the Röntgen rays should be used as they are for ringworm. The nails, if affected, may be treated in the same way as when they are diseased by the microsporon or trichophyton. The disease is very obstinate, and after apparent cure often breaks out again. It is well to care for the general health by good food and tonic medicines.

ANIMAL PARASITES

SCABIES

Scabies, or itch, is a multiform disease of the skin, consisting of papules, vesicles, pustules, and sometimes bullæ, due to the irritation of the itch-acarus, *Sarcoptes hominis*.

The female acarus is oval in shape, $\frac{1}{70}$ inch in length, presents in front four little nipple-shaped processes provided with suckers on stalks, and behind four similar processes provided with long bristles. The male is smaller, has four suckers in front, two suckers and two bristles behind. The female after impregnation bores her way under the skin in an oblique direction, so that as the superficial layers of the epidermis are detached by friction, she still remains the same depth from the surface. As she proceeds she lays her eggs, one or two daily, it is said, and she may thus burrow through the skin in an irregular line for a third or half an inch. Such a burrow (*cuniculus*), or "run," may be recognised on the surface of the skin by the following features: At one end the epidermis is broken or frayed, and the free edges are dirty; at the other end is a minute white pointed elevation, in which the acarus lies; the burrow itself between these points is a sinuous black line. The whole burrow may be snipped off with a pair of scissors curved on the flat, or shaved off with a scalpel; and if it be then moistened with liq. potassæ and examined, there will be seen the female acarus, and behind her, filling the burrow, her eggs in every stage of incubation, with minute black spots of excremental matter among them. As the skin desquamates, the most developed ova come to the surface, and are hatched. The male does not burrow, but remains on the surface, where he may be sometimes accidentally caught.

Symptoms.—As a result of the invasion of itch-acarus, there is considerable itching, with consequent scratching, pus-infection, and dermatitis of variable extent and character. The itching is mild or severe, but not generally so bad as that caused by pediculi, and the scratching rarely leaves scars or causes pigmentation. It is worse at night when the patient is warm in bed. The dermatitis consists of papules, vesicles, pustules, or even bullæ, which generally appear in the neighbourhood of the burrows, but also in parts more remote. Not infrequently a vesicle or pustule forms in the burrow itself. In addition to these lesions, patches of eczema, impetiginous crusts, and urticarious wheals are often present. There is, thus, very great variety in the lesions in different cases. In some, the burrows are numerous, with few inflammatory lesions; in others, vesicles and pustules are abundant, and burrows are with difficulty found. In some instances there are more papules, in others more pustules. The parts of the body especially liable to the attacks of the acarus

are the skin between the fingers, the front or inner side of the wrist, the front of the forearm, the ankle and foot, the axilla, the groin, and the genitals, the inner side of the thigh and the nates; and the eruption spreads beyond these parts, on to the abdomen from the groin, or along the inner side of the leg or thigh. The back, shoulders, and chest are but little affected; and the face, neck, ears, and scalp nearly always escape, except in infants at the breast, who may be infected from the mother. The occupation of the patient may have influence upon the localisation. Hebra was in the habit of recognising the occupation of a cobbler by the predominance of itch-lesions on the buttock. If the employment involves the immersion of the hands in materials (oily or otherwise) antagonistic to the acari, their presence in this typical situation will, of course, be prevented. In children, the lesions are more widespread, the feet and ankles are commonly affected, and pustules are frequently present.

Diagnosis.—The position of the lesions is an important guide to diagnosis. An itching eruption of mixed papules, vesicles, and pustules, occurring mainly about the fingers and wrists, and also in the other situations mentioned, should lead to a careful search. If a burrow can be found, the minute white elevation at the cleaner end should be looked for, and its epidermis carefully scratched through with the point of a needle; the acarus may then be picked out, as it readily adheres to the surface of the needle. If this cannot be done, it is best to snip off the whole burrow, and to examine for ova or fragments of the acarus under the microscope. Finally, where there has been much inflammation, so that burrows cannot be found, the crusts may be removed, boiled in a solution of potash or soda, the fluid allowed to settle in a conical glass, and the sediment examined for fragments of acarus.

Treatment.—Itch is generally quickly cured by the free inunction of sulphur ointment. This should be done at night, and the ointment should be left on, covered by suitable clothing, until the morning, when it may be washed off in a hot bath. The same process may be repeated on two successive nights. The disagreeable odour of sulphur ointment may be lessened by the addition of balsam of Peru (℥j to ℥j). For some skins, sulphur ointment is too irritating, and requires dilution. Storax (styracis prep. ℥ij, adipis ℥j) is also a more pleasant, less irritating, and yet effectual preparation. Vlemingkx's solution may also be used, or sulphur baths (potassium sulphide 4 ounces in 30 gallons of water). It must be remembered that the itching may continue for some time after the acarus has been destroyed. In order to prevent the recurrence of the disease, it is necessary that new clothes should be worn, and that the old ones should be quite disinfected by baking before being worn again.

PHTHEIRIASIS

(*Pediculosis*)

The pediculi or lice which infest the human race are of three species : *Pediculus capitis,* or head louse ; *P. corporis, vel vestimentorum,* or body louse ; and *P. pubis,* or crab-louse.

PEDICULUS CAPITIS

The head louse is about 2 mm. long by 1 broad, and breeds amongst the hairs of the scalp. Its ova are found adherent to the hairs, and are called nits. They are about $\frac{1}{2}$ mm. in length, whitish, somewhat conical in shape, with the apex always towards the scalp ; and they are fixed to the hair by a cylindrical sheath of chitinous material, extending some little distance beyond the apex. The irritation of the pediculus leads to constant scratching and pustular eczema, or contagious impetigo. This eruption is most common and severe in the occipital region, and the sub-occipital glands are mostly enlarged as a consequence, and may suppurate.

The **Diagnosis** is not difficult. If the pediculi are not at once seen, the nits, which are readily distinguished on careful examination from scurf, will show at once that there are or have been pediculi. The position of the crusts at the back of the head is also strongly in favour of pediculi.

Treatment.—The insects can generally at once be destroyed by the use of ung. hydrargyri ammoniati ; but if there is much eczema or impetigo the hair should be cut over it, and the crusts removed. Paraffin oil may be lightly rubbed in, and rapidly kills the animals. The nits are not easily detached from the hairs ; the cement is very resistant to acids, alkalies, and spirit, but dilute acetic acid (1 in 4) is said to soften it. They can always be slid off the hair, or may be sometimes combed off ; but if very numerous, it is probably best to cut the hair.

PEDICULUS VESTIMENTORUM

This species is larger than the head louse, being from 2 to 3 mm. long, and from 1 to $1\frac{1}{2}$ broad. It only occurs on the parts covered with clothes, and chiefly about the back and front of the chest, loins and abdomen. Occasionally the upper arms, thighs, and even legs may be attacked, but never the face or the hands. The body louse causes a severe prurigo, with intense itching, which leads to proportionately violent scratching. All the lesions described under Prurigo (*see* p. 1048) may occur, papules, blood-crusts, scratch-marks and the elongated scars which result from them, and after a time, intense pigmentation.

It occurs especially in old people, amongst the poorer classes, who have been allowed, from want of proper attention, to lapse

into conditions of filth and neglect. The disease has then been called *prurigo senilis,* and also *vagabonds' disease.*

In its milder form it presents only scattered papules, blood-crusts, and scratch-marks over the upper part of the back and shoulders.

Diagnosis.—A pruriginous eruption of this kind over the back and shoulders should always suggest a search for the pediculus vestimentorum. It is commonly found in the "gathers" under the neck-band of the shirt, or under the shelter of any edge projecting on the inner side; and it is recognised by its long oval shape and its gray colour, with a central dark-red or black spot.

Treatment.—Ung. hydrarg. ammoniati or ung. staphysagriæ, smeared over the skin, will kill the pediculi. The clothes, in which the eggs are certainly incubating, should be completely changed; and they must be baked if they are to be worn again.

PEDICULUS PUBIS

The crab-louse is smaller than either of the other species, measuring from 1 to 1½ mm. long, and from 1 to 1½ broad. It has an almost square body, and six long legs, with claws by which it clings firmly to the hairs of the part. It is not only found in the pubic hair, but is occasionally conveyed to the eyebrows, eyelashes, whiskers, or beard. The eggs are attached to the hairs close to the skin. Itching leads to scratching, and an eczematous rash is the result. Removal of the hair quickly cures the trouble; or the pediculi may be killed by the means found effectual with other species.

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